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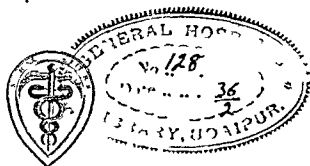
By

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M A , D M (Oxon) , F R C P , D P H (Lond )  
*Physician to the Middlesex Hospital , Physician  
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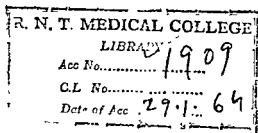
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## PREFACE TO THE FOURTH EDITION

THIS book has been carefully revised for its fourth edition, and many alterations and additions made. For convenience of reference these may be listed in the order in which they appear. New articles have been written on the following subjects: *Icterus gravis neonatorum* with erythroblastosis, epidemic catarrhal jaundice, the chronic pneumonias, adenoma of bronchus, chronic miliary tuberculosis, hæmorrhagic concussion of the lungs (blast injury of the lungs), paroxysmal flutter of the diaphragm, gastro colic fistula, non penetrating injuries of the heart, thrombosis of the axillary vein, prolapsed intervertebral disc, intracranial aneurysms, cerebral concussion, the crush syndrome, Hand Schüller Christian disease, epidemic myalgia, familial periodic paralysis, *onchocerciasis*, frost bite and poison gases.

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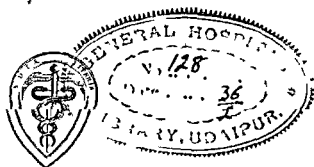
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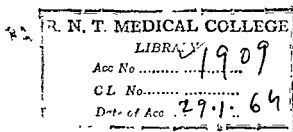
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graphy, acute subdural hæmatoma, the Guillain-Barré syndrome, spinal arteriosclerosis, automatic tidal drainage of the bladder, jelly nystagmus, progressive hypertrophic neuritis, the use of dried serum or plasma, estimation of prothrombin in plasma, hæmorrhagic thrombocythæmia, thrombocytosis, sternal puncture, blood grouping, sub-acute leukaemia, quinine in the treatment of myotonia congenita and myotonia atrophica, jaundice in glandular fever, pituitary diabetes mellitus, steatopygy, local panatropy, exophthalmic ophthalmoplegia, masked hypothyroidism, and the Laurence-Moon-Biedl syndrome.

Other additions include the therapeutic use of: Nicotinic acid, riboflavin, Sulphathiazole, Sulphadiazine, Sulphaguanidine, vitamins B<sub>6</sub>, E, K and P, Bilein, Doryl, heparin, sodium diphenyl hydantionate, Ephynal, calcium mandelate, acetylphenylhydrazine, Veritol, pamaquin, suramin, Neostam, stibophen, Caprokol, gentian violet, picrotoxin, and tetanus toxoid.

Two figures and one illustration have been added, and six figures omitted.

Minor alterations include a revision of the nomenclature of the bacteria and worms. In accordance with the suggestions made in the *British Pharmacopœia*, doses of solids have been expressed as grains and ounces, and of fluids as minims and fluid ounces. The following abbreviations have been adopted for therapeutic measurements: Grain = gr. Ounce = oz. Minim = m. Fluid ounce = fl. oz. Kilogramme = kg. Gramme = G. Milligramme = mg. Millilitre (cubic centimetre) = mil.

Proprietary preparations are distinguished by a capital letter, and the similar B.P. preparation, when available, is included in brackets. A capital letter is also used at the beginning of a prescription.

I wish to thank Major A. Willcox, R.A.M.C., and Dr. R. Wyburn-Mason for their help in reading through the sections on Heart Failure and Cerebral Aneurysms, respectively. Dr. Geoffrey Cohen kindly took the photograph for Fig. 48. I should like to take this opportunity of expressing my thanks for the letters of encouragement which I have received, and for the numerous suggestions as to alterations and additions to the text. All of these have been carefully considered and very many adopted.

The way of the publisher under present conditions is beset with difficulties and hemmed in with restrictions. Despite this, Messrs. J. & A. Churchill have never failed to smooth the author's path and render tolerable his task.

G. E. BEAUMONT.

## PREFACE TO THE FIRST EDITION

It has been my endeavour to produce a text book of medicine which is not too long for the use of the student preparing for his final examination and which will also be of assistance to the general practitioner. The ideal aimed at has been to include the essentials of medicine and to omit all extraneous matter, to give the student a clear account of the essential features of each disease described, and to supply the practitioner with information as to the investigations required to establish the diagnosis in any particular disease, together with an up to date account of a definite line of treatment.

Special attention is directed to clinical findings. Several detailed diet sheets are given, such as those suitable for the treatment of pneumonia, typhoid fever, diabetes, nephritis, gastric ulcer, obesity, constipation etc. The appropriate dosage, prescriptions and methods of administration of drugs are included in the treatment sections of the various diseases, over one hundred prescriptions being given in full. No effort has been spared to bring every article up to date, to illustrate them with explanatory diagrams, figures and temperature charts, and to connect them with cross references. A series of diagrams, illustrating the anatomy and physiology of the parts concerned, has been introduced into the chapter dealing with nervous diseases, so that this difficult branch of medicine may be more easily understood. The old anatomical terminology has been employed, but a glossary showing the corresponding terms in the international (B N A) nomenclature is included at the beginning of the book.

*It is still the duty of the general physician, attached to the teaching staff of a general hospital, to care for patients suffering from most branches of medical diseases, and to instruct students in the symptoms, signs and treatment of such diseases. Such is the reply, if reply be needed, to the criticism that the day of the one man text book has passed. If this is so, it would be argued that no single physician should be allowed to teach general medicine and have charge of general medical wards.*

Psychological and Dermatological medicine have not been included. They are highly specialised subjects, which are not dealt with in the general medical wards of a hospital. It is true that the student and practitioner must have a working knowledge of these branches of medicine, but this is best acquired from practical experience in the special departments of a general hospital. Infectious fevers have been included, as they frequently cause difficulty in the diagnosis of other medical diseases, and they are so important in general practice.

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# ANATOMICAL GLOSSARY

Old Nomenclature	International (B.N.A.) Nomenclature
Fissure of Rolando . . . . .	Central sulcus.
Sylvian fissure . . . . .	Lateral cerebral fissure.
Lenticular nucleus . . . . .	Lentiform nucleus.
Foramen of Monro . . . . .	Interventricular foramen.
Foramen of Majendie . . . . .	Medial aperture.
Foramen of Luschka . . . . .	Lateral aperture.
Sylvian aqueduct . . . . .	Cerebral aqueduct.
Valve of Vieussens . . . . .	Anterior medullary velum.
Crus cerebri . . . . .	Cerebral peduncle.
Superior corpus quadrigeminum . . . . .	Superior colliculus.
Inferior corpus quadrigeminum . . . . .	Inferior colliculus.
Superior cerebellar peduncle . . . . .	Brachium conjunctivum.
Middle cerebellar peduncle . . . . .	Brachium pontis.
Inferior cerebellar peduncle . . . . .	Restiform body.
Cisterna magna . . . . .	Cisterna cerebello-medullaris.
Cisterna basalis . . . . .	Cisterna interpeduncularis
Superior longitudinal sinus . . . . .	Superior sagittal sinus.
Inferior longitudinal sinus . . . . .	Inferior sagittal sinus.
Circular sinus . . . . .	Intercavernous plexus.
Basilar sinus . . . . .	Basilar plexus.
Lateral sinus . . . . .	Transverse sinus.
Pacchionian bodies . . . . .	Arachnoidal granulations.
Gasserian ganglion . . . . .	Semilunar ganglion.
Ganglion of Scarpa . . . . .	Vestibular ganglion.
Deiters' nucleus . . . . .	Lateral vestibular nucleus.
Column of Goll . . . . .	Funiculus gracilis.
Column of Burdach . . . . .	Funiculus cuneatus.
Direct pyramidal tract . . . . .	Anterior cerebro-spinal tract.
Crossed pyramidal tract . . . . .	Lateral cerebro-spinal tract.
Direct cerebellar tract . . . . .	Posterior spino-cerebellar tract.
Indirect cerebellar tract . . . . .	Anterior spino-cerebellar tract.
Clarke's column . . . . .	Dorsal nucleus.
Circumflex nerve . . . . .	Axillary nerve.
Lesser internal cutaneous nerve . . . . .	Medial cutaneous nerve.
Intercosto-humeral nerve . . . . .	Intercosto-brachial nerve.
External cutaneous nerve (arm) . . . . .	Posterior cutaneous nerve.
Internal cutaneous nerve (arm) . . . . .	Medial cutaneous nerve.
Musculo-spiral nerve . . . . .	Radial nerve.
Posterior interosseous nerve . . . . .	Deep branch of radial nerve.
Radial nerve . . . . .	Superficial branch of radial nerve.
External cutaneous nerve (leg) . . . . .	Lateral cutaneous nerve.
Internal cutaneous nerve (leg) . . . . .	Medial cutaneous nerve.
Middle cutaneous nerve (leg) . . . . .	Intermediate cutaneous nerve.
Small sciatic nerve . . . . .	Posterior cutaneous nerve.
Peroneal nerve . . . . .	Common peroneal nerve.
Anterior crural nerve . . . . .	Femoral nerve.
Genito-crural nerve . . . . .	Genito-femoral nerve.
Internal saphenous nerve . . . . .	Saphenous nerve.
Internal popliteal nerve . . . . .	Tibial nerve.
Anterior tibial nerve . . . . .	Deep peroneal nerve.
External saphenous nerve . . . . .	Sural nerve.
Musculo-cutaneous nerve . . . . .	Superficial peroneal nerve.
Internal plantar nerve . . . . .	Medial plantar nerve.
External plantar nerve . . . . .	Lateral plantar nerve.

# ESSENTIALS OF MEDICINE

## CHAPTER I

### THE ALIMENTARY SYSTEM

**Introductory** Special investigations are required in the elucidation of many of the diseases of the alimentary system. These include test meals, opaque meals and enemata, gastroscopy, tests for pancreatic and hepatic efficiency, cholecystography, and bacteriological and chemical examination of the feces.

### THE MOUTH AND PHARYNX

#### Gingivitis

**Definition** Inflammation of the gums. There are three varieties: Marginal, general and ulcerative. These will be considered separately.

#### Marginal Gingivitis

**Etiology** Marginal gingivitis is associated with mouth breathing, lack of efficient mastication and cleanliness of the gums, and the use of hard tooth brushes and tooth picks.

**Clinical Findings** The patient may complain of bleeding or soreness of the gums on brushing the teeth. The gums are red and swollen at their margins, or they may be retracted around the teeth. Pus may be squeezed from between the gums and teeth.

**Treatment** The causes of mouth breathing should be eradicated if possible. Tartar should be removed from the teeth and the gums massaged with the fingers towards the teeth night and morning. The tooth brush should be small and soft, and no gritty powder used. A mouth wash of milk of magnesia should be used at night.

#### General Gingivitis

**Etiology** General gingivitis may be caused by drugs such as mercury or lead, or result from ill fitting dentures and inattention to the teeth. It may also occur during pregnancy, in scurvy, or in association with any severe illness.

**Clinical Findings** The gums generally are red, swollen, sore and bleed on pressure. A blue line may be seen in lead poisoning (see p. 739).

**Treatment** This is as for marginal gingivitis. In scorbutic or pre-scorbutic conditions, as judged by urine tests, vitamin C should be given until the patient is saturated. The initial dose is ascorbic acid tab (B.P. Add.) mg. 50 2 t i d followed after a few days by mg. 50 b i d.

"comforters" forbidden. Glycerinum boracis should be sparingly applied on gauze to the patches after each feed (boric acid poisoning may occur in an infant if excessive amounts are used). Alternatively, a 1% aqueous solution of gentian violet may be applied on gauze t.i.d. for 3 or 4 days. Treatment in adults is mainly that necessary for the debilitating condition.

### Gangrenous Stomatitis (*Cancerum Oris. Noma*)

**Etiology.** The treponeme and fusiform bacillus of Vincent are usually present.

**Clinical Findings.** The patient is usually a child who is very debilitated owing to measles, diphtheria, scarlet fever or whooping-cough. Pain is noticed in the mouth, and an ulcer is seen on the inner side of the cheek which may rapidly spread and perforate the cheek. There is œdema of the face, and the gums or jaw may be similarly affected. The temperature is usually high, and death frequently follows from toxæmia or bronchopneumonia.

**Treatment.** An intramuscular injection of sulpharsphenamine should be given in a dose appropriate to the age of the child (see p. 568)

### Vesicular Stomatitis

**Etiology.** This may be a manifestation of herpes, when it is known as herpetic stomatitis or herpes buccalis. The cause is usually unknown, but herpes buccalis may occur after an injection of Sanocrysin (aur. et sod. thiosulph.) or in association with trench fever, the patient complaining that the palate burns as if irritated by a flame. The herpetic eruption may be limited to half of the hard palate or involve the tongue, gums and cheek on one or both sides. There is malaise, and pain both inside and outside the mouth, especially on mastication. The vesicles are situated on an inflamed base of mucous membrane.

In foot and mouth disease, which rarely affects man, vesicular stomatitis (epizootic stomatitis) may occur.

**Treatment.** A mouth-wash of glycerin. thymol. co. (B.P.C.) should be used.

### Ludwig's Angina

This is an acute infection of the floor of the mouth due to the streptococcus. There is a hard brawny swelling under the jaw and in the neck, and the patient is very ill. Treatment consists in free and deep incisions.

### Tonsillitis (*Amygdalitis*)

**Definition.** Inflammation of the tonsils.

The following varieties are described:—Acute, including acute follicular tonsillitis and Vincent's angina. Chronic tonsillitis.

### Acute Follicular Tonsillitis

**Etiology.** There is infection of the tonsils with pyogenic organisms, usually streptococci. The condition may be primary, or develop in

association with such diseases as rheumatic fever, acute nephritis, scarlet fever or secondary syphilis

**Pathology** The tonsils are swollen and an exudate collects in the follicles

**Clinical Findings** The patient is often a child or young adult who complains of malaise, with pain in the throat made worse by swallowing

**On Examination** The temperature is usually raised to 101° F or more, the tonsils are red, enlarged, and yellowish white spots may be seen on them, due to the exudation in the follicles. This exudation may coalesce and form a membrane, which, however, is usually limited to the tonsils and does not spread to the pharynx or uvula. The cervical glands are generally only slightly enlarged. A swab should be examined to exclude the presence of diphtheria bacilli or the organisms of Vincent's angina

**Differential Diagnosis** It is important to exclude diphtheria (see p 536), Vincent's angina, the onset of scarlet fever and the presence of active syphilis

**Course and Complications** The disease usually lasts 7 to 10 days. Nephritis may occur as a complication, and the urine should always be tested for protein and blood. Infection may spread to the cranial sinuses or middle ear, or the cervical glands may become chronically enlarged. Septicæmia and toxic myocarditis are rare but important complications. Recurrences are common especially in the early or late winter

**Prognosis** This is usually good. Repeated attacks lead to small fibrosed and cryptic tonsils, which may become foci of infection for quinsy or other diseases, such as rheumatism sciatica, or endocarditis. Death may occur from septicæmia or myocarditis

**Treatment** The patient should be put to bed kept on a liquid or semi solid diet, and the bowels opened with calomel gr 1 to 3 nocte followed by a morning dose of mag sulph gr 60 to 120. Local treatment. Hot gargles of potassium permanganate (1 in 8 000) should be used frequently, if possible. If the patient cannot gargle, the throat may be sprayed with a hot alkaline lotion such as Sod bicarb, sod benzoat., sod chlorid aa gr 10 aq ad fl oz 1 fl oz  $\frac{1}{2}$  to be used in a tumblerful of warm water occasionally, or the throat may be painted with Salol gr 30, glycerin m 120 sp vin rect ad fl oz 1. By mouth a mixture can be given, such as Sod salicyl gr 10, sod bicarb gr 20, pot chlorat gr 5, dextros gr 30, aq ad fl oz 1 Fl oz 1 tds p c Sulphanilamide 0.5 G tab, 1 to 3 tds is of value in hemolytic streptococcal infections. It should be given for 5 to 7 days. It is not now considered necessary to eliminate drugs or food containing sulphur during the administration of the sulphonamide preparations

During convalescence an iron and arsenic tonic is required, such as Liq ferri perchlor m 10 liq arsenical m 2, glycerin m 30, aq ad fl oz  $\frac{1}{2}$  17 oz  $\frac{1}{2}$  tds p c. If recurrences are frequent the question of tonsillectomy should be considered

### Vincent's Angina

**Etiology.** Ulceration of the tonsil, due to infection with the *B. fusiformis* and the *Treponema vincenti*.

**Clinical Findings.** The patient complains of symptoms resembling those of subacute tonsillitis. There is not usually severe pain and the temperature is normal or slightly raised. In some cases the patient is severely ill with high fever and muscle and joint pains.

**On Examination:** A yellowish-white membrane or ulcer may be seen on or behind one tonsil. A swab should be examined for the presence of diphtheria bacilli and Vincent's organisms.

**Differential Diagnosis.** Acute tonsillitis, diphtheria, rheumatic fever, syphilis and agranulocytic angina (see p. 506) must be excluded.

**Course.** This is benign, the condition usually clearing up spontaneously in about a week, but some cases prove more intractable.

**Treatment.** The tonsil should be treated by the application of hydrogen peroxide (10 vols.), after removal of the membrane. Liquor iodi mitis is then applied. Nicotinic acid tab. (B.P. Add.) mg. 50, 1 five times a day should be given by mouth for 7 to 10 days. If this does not prove efficacious, a solution of 0.3 G. arspenamine (B.P.C.) in 10 mls of glycerin may be used, or one injection of 0.3 G. neoarsphenamine given intravenously.

### Chronic Tonsillitis

**Etiology.** Chronic tonsillitis may result from acute tonsillitis or develop insidiously.

**Clinical Findings.** The patient is usually a child or young adult who gives a history of repeated attacks of sore throat, generally in the winter.

**On Examination:** The tonsils usually look "unhealthy," being sometimes large and almost meeting in the mid-line, or else of normal size but showing depressions or pits, or small and scarred. Pus or cheesy exudate, of very offensive odour, may in some cases be squeezed out from the tonsils. The adenoid tissue at the back of the nose may be hypertrophied. The glands in the neck may be enlarged, especially below the angle of the jaw. If there is marked obstruction to respiration and the patient is a mouth-breather, he shows the characteristic appearances; thus the lips are dry and cracked, the central teeth prominent, the gums are dry, and marginal gingivitis may be present. Further, the palate is often narrow with a high arch, and the nose is thin with feebly developed alar nasal cartilages. The chest may be pigeon-breasted. There is often some deafness, restlessness at night, and the child is backward at school.

**Course and Complications.** The patient is liable to colds, sore throats, otitis media, bronchitis and general ill-health. Local or systemic infections, such as diphtheria, scarlet fever, rheumatism, etc., may occur in association with chronic tonsillar sepsis.

**Prognosis.** This is favourable, as the disease can be eradicated surgically.

**Treatment.** In the majority of cases the tonsils and adenoids

should be removed by operation. Medical treatment consists in the use of a throat paint such as the *Pigmentum Mandi* (Iodin gr 6 pot iod gr 20 ol menth pip m 5 glycerin ad fl oz 1)

*The Lingual Tonsil* This is situated at the base of the tongue, and when enlarged may cause a persistent, irritating cough curable by operation

### Quincy

#### (*Peritonsillar Suppuration Peritonsillitis*)

**Definition** Abscess formation in the connective tissue around the tonsil

**Etiology** Quincy is generally secondary to chronic tonsillitis

**Clinical Findings** The patient, who is usually a young adult, feels ill with shooting pains in the ear and throat, rigors often intense dysphagia and inability to open the mouth fully

**On Examination** The breath is offensive and the mouth is kept slightly open. A swelling due to the abscess is seen usually above and on the outer side of one or other tonsil. This pushes the tonsil out of view and the uvula is deviated from the mid line

**Differential Diagnosis** A sarcoma of the tonsil may be mistaken for quincy, with the former there is usually no fever

**Course and Complications** The abscess points in a few days and usually bursts through the anterior pillar of the fauces with immediate relief of pain. Complications are rare. They include suppuration in the neck, pyæmia, hæmorrhage œdema of the glottis thrombosis of the internal jugular vein or even asphyxia from inhalation of the pus if the abscess ruptures during sleep. Blockage of a few bronchi by inhaled pus will lead to collapse of a portion of the lung and hæmoptysis may result from the inflamed bronchial mucous membrane

**Treatment** In the early stages the treatment is as for acute follicular tonsillitis (see p 5). The attack is best terminated surgically by incision as soon as pus has formed and the tonsils should be removed later

### Tuberculosis of the Tonsils

**Pathology** The tonsil may very rarely be affected clinically apart from pulmonary tuberculosis. The patient complains of a chronic sore throat and generally of symptoms of pulmonary tuberculosis, such as cough, expectoration malaise etc

**On Examination** The tonsil may be ulcerated. In the majority of cases there is definite pulmonary and laryngeal tuberculosis and tubercle bacilli are present in the sputum. In another group of cases the clinical features are those of cervical adenitis the tubercle bacilli gaining entrance through the tonsils and settling in the glands in the neck. In such cases tubercles may or may not be present macroscopically in the tonsils but tonsillectomy is usually advisable

### Syphilis of the Tonsils

The tonsil may be infected in the primary stage when the lesion present is a chancre. In secondary syphilis a sore throat with acute

tonsillitis or the formation of superficial plaques (snail-track ulceration) on the tonsils may occur. In tertiary syphilis ulceration due to a gumma may be noted.

### Tumours of the Tonsil

These are nearly always malignant, being either an epithelioma or sarcoma (lymphosarcoma or round-celled sarcoma). In epithelioma there is usually ulceration of the tonsil and hard enlarged glands in the neck. In lymphosarcoma the tonsil appears swollen, pale and waxy, or may be ulcerated. It grows rapidly, and neighbouring glands are usually soon enlarged.

### The Tongue

An examination of the tongue does not give so much information concerning the health of the patient as was at one time believed. Thus, the tongue may be almost black (melanoglossia) apart from taking drugs, and yet the patient be in good health, or it may be clean and moist and the patient be suffering from a mortal illness such as tuberculosis.

*Furred Tongue.* A white or brown coat may result from a milk diet or from drugs such as bismuth or iron, or it may be associated with fevers, chronic alcoholism, gastro-intestinal disorders, or local causes such as carious teeth.

*A soft flabby tongue* with impressions of the teeth on its edges may occur in atonic dyspepsia and gastric hyposecretion.

*A red firm tongue* may be associated with gastric hypersecretion or with diabetes mellitus (raw beef tongue).

*A fissured tongue* (scrotal tongue) occurs usually as a congenital condition, and is of no pathological significance. In tertiary syphilis the tongue may be fissured.

*A white strawberry tongue* occurs in scarlet fever. The papillæ are covered with a white fur.

*A red strawberry or raspberry tongue* occurs in a later stage of scarlet fever. The tongue has peeled and the bright red papillæ stand out.

*A dry glazed or brown tongue* occurs in the terminal stages of severe illnesses, such as cholera, dysentery and septicæmia.

*A slaty-blue tongue* may be seen in Addison's disease.

*A black tongue* (melanoglossia). The posterior portion of the tongue around the circumvallate papillæ is dark-brown or black. A fungus, *aspergillus niger*, may be present in the scrapings, but is probably not pathological. There is usually no digestive trouble and the tongue eventually becomes normal.

*A sore tongue* may occur in ulceration from any cause (see p. 10), in the early stages of pernicious anæmia, in sprue or in mercurial poisoning.

*A smooth glazed tongue* may be seen in pernicious or simple achlorhydric anæmia.

### Glossitis

**Definition.** Inflammation of the tongue. There are two varieties: Acute and chronic glossitis.

## Acute Glossitis

**Etiology** Acute glossitis may result from abrasions of the tongue associated with dental caries, or from insect bites or burns. Occasionally acute glossitis complicates severe fevers or small pox. The infection is usually streptococcal.

**Clinical Findings** The patient complains of pain and swelling of the tongue and the pain may radiate to the ears. There is usually marked prostration and high fever.

**On Examination** The tongue is red and swollen, and enlarged glands may be felt in the neck.

**Course and Complications** The disease is usually rapidly progressive. Abscesses or gangrene may occur in the tongue, and complications include œdema of the larynx, Ludwig's angina and septicæmia.

**Prognosis** This is very unfavourable, the majority of cases proving fatal.

**Treatment** Cold applications such as ice should be placed on the tongue. One mil of antistreptococcal serum may be injected locally, after a superficial longitudinal incision has been made into the tongue. Sulphanilamide, 0.5 G tab, 1 to 3 t i d for 3 to 4 days should be given for hæmolytic streptococcal infections.

## Chronic Glossitis

**Etiology** Chronic glossitis may result from irritation due to smoking, from syphilis, chronic alcoholism or occur in association with oral sepsis, anemia and achlorhydria. A raw, glazed and superficially fissured tongue, often associated with cracked leukoplakic angles of the mouth (angular stomatitis) may be met with in pellagra, tropical sprue, pernicious anemia, the nutritional anemias and idiopathic steatorrhea. It is thought to be due to deficiency of the vitamin B complex.

**Clinical Findings** The patient complains of a sore tongue, and in severe cases the pain may keep him awake.

**On Examination** Red smooth patches may be seen on the tongue.

**Course and Complications** The condition is usually of long duration, and leukoplakia may develop.

**Prognosis** This is usually good if adequate treatment is begun early.

**Treatment** All irritating food should be avoided, smoking and alcohol must be forbidden. Any septic focus in the mouth or a sharp tooth should be treated. Tincture of hamamelis in 60 in water fl oz 1 may be applied to the tongue night and morning. Nicotinic acid tab (B.P. Add.) mg 50, 1 or 2 t i d should be given by mouth for the glossitis due to vitamin B<sub>3</sub> deficiency and for the angular stomatitis. Riboflavin tab mg 1 t d s.

## Leukoplakia Buccalis

**Definition.** A condition of keratosis of the tongue or mucous membrane of the mouth with thickening of the deeper tissues.



**Etiology.** Leukoplakia is usually associated with syphilis, over-smoking, chronic alcoholism and oral sepsis.

**Clinical Findings.** The patient is commonly a male over the age of 40. There are usually no symptoms, but thickened whitish-grey patches occur on the tongue, and at times on the cheeks or gums.

**Course and Complications.** Ulceration or epitheliomatous changes are prone to occur.

**Treatment.** Owing to the risk of malignant disease, excision of the patches is usually recommended. Anti-syphilitic treatment is generally of no avail.

### Geographical Tongue

#### *(Eczema of the Tongue)*

**Etiology.** The cause is unknown.

**Clinical Findings.** The patient may complain of some itching or burning of the tongue.

**On Examination :** Whitish rings may be seen surrounding a red area of the tongue. The rings may spread and fuse with each other forming outlines resembling a map. Fresh lesions begin as small, whitish patches which shed their epithelium at the centre.

**Course.** The condition generally persists for some time.

**Treatment.** A 1% silver nitrate solution may be applied twice a week to relieve burning.

### Ulcers of the Tongue

These may be: 1. Simple, due usually to irritation of a tooth, "dyspeptic," or associated with ulcerative stomatitis. 2. Granulomatous, caused by syphilis or tuberculosis. 3. Malignant, an epithelioma.

### Tumours of the Tongue

These are simple or malignant. A papilloma may occur, sessile or pedunculated. Operative removal is always advisable. An epithelioma is usually found in men over the age of 40. It may develop from a papilloma or from a leukoplakic patch. There is a tendency to ulceration and secondary deposits are found in the cervical glands. Treatment is surgical, either by radium, diathermy or excision.

### Granulomata of the Tongue

A gumma forms a firm slightly raised swelling, often seated near the centre of the tongue.

Tuberculosis causes painful ulceration, frequently near the tip, and is associated generally with pulmonary tuberculosis.

## THE PHARYNX

### Pharyngitis

**Definition.** Inflammation of the pharyngeal mucous membrane.

There are two varieties: Acute and chronic. Acute pharyngitis may be catarrhal or septic.

**Acute Catarrhal Pharyngitis***(Sore Throat)*

**Etiology** Acute catarrhal pharyngitis may be due to a cold or occur at the onset of specific fevers, such as measles or scarlet fever, or be caused by drugs, such as iodides or mercury. It is also sometimes seen in the secondary stage of syphilis or it may follow tonsillitis.

**Clinical Findings.** The patient complains of rawness at the back of the throat, with perhaps dysphagia.

**On Examination** The pharynx is red and congested. General constitutional disturbance with slight pyrexia is usually present.

**Course and Complications** A mild form of laryngitis may follow after the pharyngitis has disappeared.

**Prognosis** This is usually good.

**Treatment** Relief is obtained by a steam inhalation containing carbolic acid m 5 in one pint of steaming water at 160° F, or by a gargle of sod bicarb gr 15, aq ad fl oz 10.

**Acute Septic Pharyngitis**

**Etiology** Acute septic pharyngitis is due to a streptococcal or a pneumococcal infection.

**Clinical Findings** The pharynx is red, oedematous or sloughing, or a grey, slimy exudate may form. The patient is very ill with a subnormal or a high temperature.

**Complications** These include oedema of the larynx, Ludwig's angina, pneumonia, parotitis and septicæmia.

**Prognosis** This is grave.

**Treatment** Steam inhalations and hot fomentations may be used. For hæmolytic streptococcal infections sulphanilamide should be given, and for pneumococcal ones Sulphapyridine (M & B 693). The dosage is 0.5 G tab, 1 to 3 t i d for 3 to 4 days.

**Chronic Pharyngitis**

There are three varieties of chronic pharyngitis. Simple or catarrhal (relaxed throat), granular or hypertrophic, atrophic (pharyngitis sicca).

**Simple or Catarrhal Pharyngitis**

Simple pharyngitis is due to over smoking, chronic alcoholism, dust or oral sepsis.

**Clinical Findings** The patient is usually an adult male who complains of rawness, a tickling or pricking sensation in the throat, or of an ineffective paroxysmal cough. The voice may be hoarse.

**On Examination** The pharyngeal mucous membrane appears congested and the venules may be dilated.

**Granular Pharyngitis**

Granular pharyngitis is also known as Clergyman's sore throat, and may occur in association with catarrhal infections or some weakness of the voice, and in children in association with adenoids.

**Clinical Findings.** The symptoms are much the same as those of simple pharyngitis.

**On Examination:** Small gelatinous-looking swellings (lymphoid nodules) are seen on the pharyngeal wall.

### Atrophic Pharyngitis

This is associated with rhinitis sicca.

**Clinical Findings.** The symptoms resemble those of simple or granular pharyngitis.

**On Examination:** The pharynx is red and shiny, and muco-pus may be seen running down from the posterior nares.

**Treatment.** In all forms of chronic pharyngitis, irritants such as tobacco and alcohol should be avoided. A throat paint of Liq. ferri perchlor. m. 60, glycerin. ad fl. oz. 1, or Mandl's paint, Iodin. gr. 6, pot. iod. gr. 20, ol. menth. pip. m. 5, glycerin. ad fl. oz. 1, should be applied night and morning. Septic foci in the mouth and nose should be treated; speakers should rest the voice and, if necessary, take lessons in voice production.

### Retropharyngeal Abscess

**Definition.** Suppuration in the submucous connective tissue behind the posterior pharyngeal wall. The prevertebral glands are first affected.

There are two varieties: Acute and chronic.

#### Acute Retropharyngeal Abscess

**Etiology.** Acute retropharyngeal abscess may occur in association with suppuration in the nose or posterior pharyngeal lymph glands, septic tonsillitis, otitis media, or as a complication of scarlet fever, diphtheria and measles.

**Clinical Findings.** The patient is usually an infant, who has pain in the throat, and difficulty in swallowing or breathing. The cry may be quacking in character (*cri de canard*) and croup sometimes occurs.

**On Examination:** The child appears ill and is feverish. A bulging of the posterior pharyngeal wall can be seen, usually not quite central. It may be possible to feel fluctuation.

**Course.** Death may occur from rupture of the abscess and suffocation.

**Treatment.** This is surgical. An incision should be made through the mouth into the posterior pharyngeal wall without an anæsthetic, the child's head being lowered, so that the pus is not inhaled.

#### Chronic Retropharyngeal Abscess

**Etiology.** The condition is usually tuberculous, secondary either to tuberculous cervical or retropharyngeal glands, or to caries of the upper cervical vertebræ.

**Clinical Findings.** The patient is usually a child, and is commonly

afebrile. There may be some dyspnoea or snoring, but often there is no pain. Enlarged tuberculous glands or signs of cervical caries may be evident, and bulging may be seen in the posterior pharyngeal wall.

**Treatment** This is surgical. The abscess should be opened externally in the neck.

### Pharyngeal Ulcers

These may be 1 Follicular 2 Syphilitic. In primary syphilis a chancre may be seen, in secondary syphilis there are mucous plaques or pharyngitis, and in the tertiary stage ulcers or gummata may occur. 3 Tuberculous. Lupus may spread from the nose to the mouth, palate and pharynx, it shows a typical apple jelly appearance. In advanced pulmonary tuberculosis there may be very painful ulcers in the pharynx. 4 Accompanying fevers, such as typhoid. 5 Epitheliomatous. 6 Diphtheritic.

### Enlargement of the Uvula

The uvula may be enlarged in patients suffering from pharyngitis, relaxed throat, nephritis with cedema, or anæmia (cedematous). It may give rise to a cough.

### Tumours of the Pharynx

These may be simple, such as a papilloma or angioma, or malignant, such as an epithelioma or sarcoma. Malignant tumours are rare.

### Pharyngeal Neuroses

1 *Globus Hystericus*. This is a motor spasm. The patient complains of a lump in the throat. The lingual tonsil may be enlarged.

2 *Anæsthesia*. This occurs especially in hysteria.

3 *Paræsthesia*. The patient complains of a feeling of suffocation and tickling in the throat.

Diverticula of the pharynx are considered later (see *Œsophagus*, p. 20).

## THE SALIVARY GLANDS

### Ptyalism

#### (Salivation)

**Definition** Over secretion of the salivary glands.

**Etiology** The most important causes are 1 Drugs especially mercury, iodides, arsenic and pilocarpine. 2 Reflex. Oral, such as dental caries, stomatitis, and dentition. Œsophageal, as with a growth. Gastric, at the onset of vomiting, gastric ulcer, etc. Hepatic and pancreatic inflammatory lesions. 3 Nervous lesions as in tabetic crises, tic douloureux, encephalitis lethargica and paralysis agitans. 4 Dysphagia due to mechanical causes as in fractured jaw, mumps etc., or due to neuro muscular causes as in bulbar paralysis, bilateral facial paralysis, myasthenia gravis and hydrophobia. In cases associated with dysphagia the saliva dribbles away and the secretion may not be excessive. Ptyalorrhœa is a functional condition, which may

complicate pregnancy or occur in association with a high blood pressure.

**Treatment.** In each case this is directed to the underlying cause, but atropine and bromides may be given such as Atropin. sulph. gr.  $\frac{1}{60}$ , aq. ad m. 60. t.d.s. a.c.; or Pot. brom. gr. 10, aq. chlorof. ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  t.d.s. p.c. X-ray treatment to the salivary glands may be required in obstinate cases.

### Xerostomia

(*Aptyalism*)

**Definition.** Dryness of the mouth.

**Etiology.** Xerostomia may be due to: Drugs, especially belladonna and opium. Fevers. Deficient fluid intake. Emotions such as fear. Local causes such as thrush or chronic inflammation of the salivary glands as in Mikulicz's disease. Old age, especially in women. In association with excessive loss of body fluids as in cholera and diabetes.

**Treatment.** Local or general causes should be treated. Acid substances such as unsweetened lime juice and a mouth-wash of glycerin and lemon juice may be useful.

### Acute Septic Parotitis

(*Parotid bubo*)

**Definition.** Acute inflammation of the parotid gland.

**Etiology.** Acute septic parotitis is usually due to infection ascending the parotid duct. The causative organism is frequently the staphylococcus aureus, less often the streptococcus viridans or the Diplococcus pneumoniae (pneumococcus). It may occur in the absence of mastication, in fevers such as enteric, in pneumonia, in facial paralysis, in cleft palate where an obturator is worn, in chronic uræmia, after operations on the abdomen, or after a trivial injury to the genital organs.

**Clinical Findings.** The patient complains of pain and swelling in the region of one parotid gland, with dysphagia and malaise.

**On Examination:** A tender parotid swelling is found with redness of the overlying skin. The patient is obviously ill.

**Course and Complications.** Suppuration usually occurs in the gland.

**Prognosis.** This is grave and death is not infrequent.

**Treatment.** The gland should be opened surgically as soon as fluctuation occurs; previously fomentations may be applied. Mouth-washes should be used frequently.

### Acute Specific Parotitis (see Mumps, p. 550)

#### Chronic Parotitis

In chronic inflammation of the parotid glands, the other salivary glands are usually also affected. This may result from such causes as drugs (iodides, mercury or lead), syphilis, mumps, calculi and chronic nephritis. Treatment is directed to the underlying cause.

### Mikulicz's Disease

**Definition.** A disease characterised by chronic swelling of the salivary and lachrymal glands.

**Etiology.** The cause of Mikulicz's disease is probably a low grade infection

**Pathology** Various types occur, varying from simple inflammation of the glands to leukæmia, lymphadenoma and lymphosarcoma Syphilitic or tuberculous changes are rarely found in the glands

**Clinical Findings** The patient complains of gradual swellings in the region of the eyes and face There is interference with vision, with some limitation of the temporal fields owing to the enlarged lachrymal glands, dryness of the eyes and mouth, but usually no pain

**On Examination** Swellings are seen in the region of the lachrymal, parotid and submaxillary glands, and there may be ptosis of the eyelids

**Differential Diagnosis** The gradual onset, absence of pain and the disposition of the swellings usually make the diagnosis clear. Uveo-parotid tuberculosis may be mistaken for Mikulicz's disease In the former there is enlargement of the parotid, submaxillary, and sometimes of the lachrymal glands, with inflammatory lesions in the uveal tract, and often facial paralysis, or more rarely polyneuritis

**Course and Complications** The course is usually chronic. Leukæmic or lymphosarcomatous changes may show themselves

**Treatment** Arsenic and iodides should be given by mouth, such as Liq arsenical m 2, pot iod gr 5, sod bicarb gr 10 sp chlorof m 5, infus gent co rec ad fl oz 1 Fl oz 1 t d s p c

If this is not successful the glands may be treated by X rays or surgical removal of the enlarged lachrymal glands may be followed by disappearance of the swelling of the salivary glands

### Tumours of the Salivary Glands

The majority of tumours of the salivary glands are mixed ones, containing myxomatous, fibrous and cartilaginous tissue, with a low grade of malignancy They should be removed surgically and the scar afterwards treated with X rays The parotid gland is usually affected

### Salivary Calculi

Calculi, rarely bilateral, form usually in the submaxillary glands or ducts They may give rise to painful swelling of the glands, especially provoked by foods which stimulate salivation or by acid substances The calculus may be palpable or may only be seen by X rays It should be removed surgically

## THE ŒSOPHAGUS

### Œsophagitis

**Definition** Inflammation of the Œsophagus This may be acute or chronic

### Acute Œsophagitis

**Etiology.** Acute Œsophagitis may be due to chemical irritants such as poisons (carbolic acid, perchloride of mercury or poison gases), to impacted foreign bodies, or it may be associated with tuberculosis, diphtheria, syphilis or carcinoma It may also occur in small pox or enteric fever

**Pathology.** In cases of poisoning the lesion is usually situated at the lower end of the œsophagus. The changes vary from hyperæmia of the mucous membrane to abscess formation. Mediastinitis is often present.

**Clinical Findings.** There may be a history of any of the causative conditions described above. The patient complains of pain on swallowing and under the sternum; vomiting of blood and mucus may occur. In severe cases there is fever with rigors and marked constitutional disturbance.

**Differential Diagnosis.** The history of the case and dysphagia with retrosternal pain usually render the diagnosis clear.

**Course and Complications.** In mild cases the course is that of rapid recovery; perforation of the œsophagus or submucous abscess formation (phlegmonous œsophagitis) may lead to mediastinitis which is quickly fatal. Lesser degrees of ulceration may result in subsequent stenosis.

**Prognosis.** This varies with the cause and the degree of inflammation. There may be complete recovery, death or subsequent stenosis.

**Treatment.** No food must be given by mouth until the patient is able to swallow a little olive oil without pain. An injection of morphin. sulph. gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  may be given during the acute stage. The possibility of subsequent stenosis should be remembered. Œsophagoscopy or an opaque meal will indicate any degree of obstruction with dilatation above it. Stricture should be prevented by the passage of bougies.

### Chronic Œsophagitis

**Etiology.** Chronic œsophagitis may be due to alcoholism, achalasia of the cardia, frequent vomiting associated with pyloric stenosis, repeated passage of a stomach tube, tuberculosis, syphilis, actinomycosis, diverticula, new growths or a cerebral tumour.

**Clinical Findings.** The symptoms of chronic œsophagitis are usually indistinguishable from those of the underlying causes, which are described later. In addition there is usually a burning sensation in the back and under the lower third of the sternum. Repeated small hæmatemeses may occur, and œsophageal spasm will cause dysphagia.

**Treatment.** The diet should be soft and bland, and the following mixture given, Sod. brom. gr. 10, tnc. belladon. m. 15, aq. chlorof. ad fl. oz. 1. Fl. oz. 1 t.d.s. In addition olive oil m. 60 to 120 should be given immediately before each feed.

### Œsophageal Obstruction

**Etiology.** The blockage may be due to: 1. Obstruction in the lumen as by impacted foreign bodies. 2. Changes in the walls (intrinsic causes) such as stenosis, congenital or acquired; the latter may be due to spasm. Other causes include fibrosis from ulceration (due to trauma, chemicals, diphtheria, new growth, or gumma) and tumours, especially carcinoma. 3. External pressure (extrinsic causes) due to aneurysm,

an enlarged thyroid, enlarged glands, mediastinal growth, pericardial or pleural effusion, pharyngeal or œsophageal diverticula, vertebral exostoses or new growths, or cervical caries

Certain of these conditions will now be described in more detail

### Congenital Atresia

The œsophagus ends in communication with the trachea or a bronchus, so that food swallowed passes into the lungs causing cough and regurgitation. Less frequently the upper portion of the œsophagus ends blindly, the lower end communicating above with the trachea or a bronchus. Excessive mucus in the pharynx is usually noted at birth. Death usually occurs a day or so after birth.

### Congenital Stenosis

The lumen of the œsophagus is narrowed, but there is no cicatrization. Clinically, regurgitation of food shows itself when the infant takes solids.

### Œsophageal Spasm

(*Œsophagismus*)

Three varieties occur —

1 *Primary Œsophageal Spasm*. This is a neurosis. The patient experiences difficulty in swallowing the bolus sticking in the gullet. Globus hystericus, hiccough or regurgitation of food may occur. The spasm is transitory and may take place at any level in the œsophagus.

**Treatment.** Cure can usually be effected by suggestion.

2 *Reflex Œsophageal Spasm*. This may occur in association with local lesions such as a growth or ulcer in the œsophagus, or with inflammation of the gall bladder or stomach.

3 *Plummer Vinson Syndrome*. This is probably caused by a disturbance of the neuro-muscular mechanism at the junction of the pharynx and œsophagus.

**Pathology.** There is absence of relaxation of the pharyngo-œsophageal sphincter.

**Clinical Findings.** The patient is often a woman about the age of 40. There is difficulty in swallowing solids and in some cases liquids, the food sticking at the back of the throat.

**On Examination.** The tongue is smooth and may be sore. The pharynx is pale and dry. The skin is pale brownish yellow, and the blood shows a microcytic hypochromic anemia (see p. 492), with increased fragility of the red cells. Achlorhydria is frequently found. The spleen and liver may be enlarged.

**Course and Complications.** The course is progressive, if untreated. Carcinoma at the junction of the pharynx and œsophagus (post-cricoid carcinoma) and pernicious anemia may occur as complications.

**Treatment.** This consists in the passage of mercury bougies (see p. 18) and the administration of ferri et ammon. cit. gr. 20 to 40 t. d. s. p. c. (see p. 492).



## Cardiospasm

(*Achalasia of the cardia. Idiopathic dilatation of the œsophagus. Phrenospasm. Hiatal œsophagismus.*)

**Etiology.** An obstruction is said to occur at the lower end of the œsophagus, due to failure of relaxation of the cardiac sphincter on deglutition, and caused by a disturbance of the neuro-muscular mechanism. There is probably no spasm of the right crus of the diaphragm, but in some cases there is spasm of the true intrinsic sphincter at the cardia. There is evidence to show that this sphincter contracts and relaxes on sympathetic and vagal stimulation respectively.

**Pathology.** The œsophagus is dilated, with inflammation of the mucous membrane and hypertrophy of the circular muscle fibres. The cardia is normal. There is degeneration of Auerbach's plexus in the cardiac sphincter in some cases.

**Clinical Findings.** The patient is usually an adult over the age of 20, of either sex. He may give a history of some discomfort on swallowing for many years, and later of regurgitation of solid food or at times of a feeling of obstruction. There is also often a choking sensation and substernal pain. This occurs usually directly after swallowing, but when much dilatation has taken place vomiting may be delayed for half an hour or so. Achalasia of the cardia may directly ensue after the vomiting of pregnancy.

**On Examination:** The patient is not usually wasted except in advanced cases, and no physical signs are found. The opaque meal shows the dilated œsophagus above, and obstruction at the level of the diaphragm.

**Differential Diagnosis.** Other causes of œsophageal obstruction must be excluded, especially carcinoma of the œsophagus, a fibrous stricture resulting from a healed ulcer, and aneurysm. The barium meal and X-ray examination may suggest the irregular outline of a carcinoma, and a fractional test meal may show a curve suggestive of carcinoma of the stomach. Thus there may be high total acidity, no free HCl and some blood, if the fermenting contents are aspirated from the dilated œsophagus. Clinically, the long history, absence of marked cachexia and wasting negative this diagnosis and œsophagoscopy excludes it.

**Course and Complications.** Achalasia usually persists and gets worse, if untreated. Hæmorrhage, the formation of a diverticulum, rupture of the œsophagus and general wasting may ensue. Carcinoma is a very rare complication.

**Prognosis.** This is favourable if proper treatment is given, especially if the condition has not existed very long.

**Treatment.** A mercury bougie should be passed, at first directly before every meal, and later at less frequent intervals. The bougie is a closed rubber tube, varying in size up to a No. 24 gauge, containing mercury. The weight of the tube causes its passage, after swallowing, down the œsophagus and into the stomach. The patient soon learns to do this for himself, and to begin with, the bougie should be left *in situ* for

ten to fifteen minutes each time it is passed. Later, it is removed as soon as it has been passed. Operation may be required in cases which do not respond to medical treatment. Good results have been obtained by the crude method of digital dilatation of the lower end of the œsophagus by opening the stomach. In other cases a lower œsophageal sympathectomy has been successful.

### Carcinoma of the Œsophagus

**Etiology.** The cause is unknown.

**Pathology.** The growth usually occurs either in the upper or lower end of the œsophagus, or where it is crossed by the left bronchus. Three types occur—an ulcerative, a scirrhus, or, more rarely, a fungating form. It may infiltrate surrounding structures in the neck or mediastinum. The growth is usually of the squamous-celled variety, but the fungating form may be a columnar celled adenocarcinoma arising from the mucous glands.

**Clinical Findings.** The patient is usually a male over the age of 40. He complains of dysphagia which may have a sudden onset, but more often comes on gradually, being first noticed on swallowing solids and later fluids. Subsequently vomiting occurs directly after swallowing, often of frothy material mixed with food. An early symptom may be substernal pain. The patient rapidly loses weight and becomes cachectic as the stenosis increases.

**On Examination.** In the early stages there are no physical signs. An X ray examination with the screen and swallowed barium meal indicates the point of stricture and excludes the presence of an aneurysm. Œsophagoscopy will definitely confirm the diagnosis. Blood may be present in the vomit or in the stools. Enlarged glands may be felt in the neck, especially above the left clavicle.

**Differential Diagnosis.** Other causes of œsophageal obstruction, such as aneurysm, achalasia and syphilitic stricture, must be excluded, as described above.

**Course and Complications.** The course is progressive. Complications such as perforation of the œsophagus, hæmorrhage and extension of the growth into the surrounding structures such as the mediastinum and lungs, and septic bronchopneumonia may occur. The involvement of the mediastinum may give rise to deficient air entry into one or other lung, or to unilateral or bilateral recurrent laryngeal paralysis.

**Prognosis.** Death is inevitable within 6 to 12 months from the onset of symptoms.

**Treatment.** This is largely palliative, an early gastrostomy may prolong life by enabling nutrition to be maintained by direct feeding into the stomach. To relieve dysphagia Souttar's tube may be used. This is "a flexible spiral formed of German silver wire and gilded." It has an expanded upper end and a twisted oval section, which prevents displacement. It is introduced through the growth with an œsophagoscope, and allows the patient to swallow solid food. Radium may be inserted into the œsophagus, or the growth treated with a radium bomb.

or by deep X-rays. Total excision is an operation which is almost invariably impossible, or, if attempted, fatal.

### Sarcoma of the Œsophagus

This is much less common than carcinoma. It may form a polypoid or ulcerating growth.

### Simple Tumours of the Œsophagus

Simple polypi may occur, giving rise to obstructive symptoms. *Œsophagoscopy enables them to be diagnosed and removed.* Other simple tumours include fibroma, fibromyoma, an accessory thyroid tumour and a simple cyst.

### Syphilis of the Œsophagus

**Etiology.** Syphilis of the œsophagus is usually due to acquired disease, rarely occurring in the congenital variety.

**Pathology.** In secondary syphilis there is inflammation of the mucous membrane. The lesions take the form of an *œsophagitis* which may give rise to dysphagia. Gummata may occur in tertiary syphilis at the upper or lower end of the œsophagus. These may ulcerate. Leukoplakia is often seen in the mouth and in the œsophagus. Obstruction is intensified by muscular spasm.

**Clinical Findings.** The patient is usually an adult of either sex who complains of progressive dysphagia. There is commonly no pain unless the pharynx is also involved. Signs of syphilis may be found elsewhere. The blood Wassermann reaction may be negative, but that of the cerebro-spinal fluid is usually positive.

**Differential Diagnosis.** This can only be made with certainty by œsophagoscopy, as a positive Wassermann reaction due to syphilis elsewhere may occur in association with a carcinoma of the œsophagus.

**Course and Complications.** Progressive stenosis usually develops if the lesion is left untreated.

**Prognosis.** This is very favourable with adequate treatment.

**Treatment.** A full course of anti-syphilitic treatment should be given (see p. 43). Stenosis should be prevented by repeated dilatation with bougies.

### Diverticula of the Œsophagus

**Definition.** Pouches formed by herniation of the œsophageal mucous membrane through the muscular coat.

**Etiology.** There are two varieties: 1. *Traction Diverticula.* These are caused by adhesions between the œsophagus and chronically inflamed tuberculous bronchial glands, at the bifurcation of the trachea. 2. *Pulsion Diverticula.* These are due to increased internal pressure associated with swallowing food and weakness of the œsophageal wall.

**Pathology.** Traction diverticula are usually small and the whole coat of the œsophagus is involved. They arise from the anterior wall, near the tracheal bifurcation. Pulsion diverticula may be small or large. They are commonly situated at the junction of the pharynx

(pharyngeal diverticula) with the œsophagus on the posterior wall at the level of the upper and lower division of the inferior constrictor muscle of the pharynx.

**Clinical Findings** Diverticula usually give rise to no symptoms unless they are sufficiently large to interfere with deglutition. Accumulations of small quantities of food may cause an unpleasant taste in the mouth, regurgitation of food may occur, or a swelling be noticed in the neck which diminishes after regurgitation causing dysphagia or cough.

**Differential Diagnosis.** A swelling in the neck which disappears after regurgitation of food is very suggestive. An X ray examination after swallowing a barium paste usually confirms the diagnosis.

**Course and Complications** A large diverticulum may cause œsophageal obstruction owing to pressure of its contents.

**Treatment** Small diverticula are usually only diagnosed by X ray examination and require no treatment. A larger diverticulum in the neck can be removed by operation. The rarer varieties in the thorax are usually intractable. Sometimes the patient can swallow more easily if he lies on his face.

### Œsophageal Varices

Varicose veins in the lower part of the œsophagus are a common *post mortem* finding. They are usually associated with cirrhosis of the liver, consequent on portal vein stagnation. They may also occur with obstruction of the inferior or superior vena cava, as in heart failure. They are a cause of hæmatemesis or melaena and can only be diagnosed during life by œsophagoscopy.

### Œsophageal Ulceration

The following varieties of ulcer may occur. **Traumatic**, peptic, simple as a complication of fevers such as diphtheria or scarlet fever, malignant, syphilitic, and tuberculous. Peptic ulcers occur at the lower end of the œsophagus. They cause pain on swallowing at the lower end of the sternum and in the back. Vomiting and hæmatemesis may occur. The diagnosis of ulceration is made by œsophagoscopy, and the treatment is as for gastric ulcer (see p. 96), but olive oil in 60 to 120 should be given immediately before each feed.

### Rupture of the Œsophagus

This may occur spontaneously when ulceration is present, or from trauma caused by an ingested foreign body or the passage of an œsophagoscope or bougie. It may rarely follow vomiting. Thus a patient after rapidly drinking several glasses of beer, may vomit violently and be seized with acute pain in the left side of the chest and left flank. He sits up and writhes in agony.

**On Examination** The abdominal wall is rigid and a pleural rub is heard over the left lower chest in front. The pulse is rapid, and the temperature subnormal. In a few hours signs of a left hydropneumothorax are present, acid stomach contents can be aspirated from the

lower left chest and air is present above. Death ensues in about 30 to 40 hours, and at autopsy a rent is found in the œsophagus just above the diaphragm.

### Dilatation of the Œsophagus

This may be a uniform dilatation occurring above an œsophageal obstruction (see Dysphagia) or be due to achalasia of the cardia. Localised dilatation occurs as a diverticulum.

### Dysphagia

**Definition.** Difficulty in swallowing.

**Etiology.** The causes may be classified as oral, pharyngeal, laryngeal and œsophageal. The most important causes are:—

**Oral.** Stomatitis; ulcers of the tongue; sore throat as in tonsillitis; quinsy; cleft palate; palatal and pharyngeal paralysis as in diphtheria, bulbar palsy, myasthenia gravis, and progressive muscular atrophy; mumps; fractured jaw; arthritis of the jaw; dislocated jaw.

**Pharyngeal.** Retropharyngeal abscess; diverticulum; syphilitic stenosis; achalasia of the pharyngo-œsophageal sphincter (Plummer-Vinson syndrome).

**Laryngeal.** Tuberculous laryngitis; carcinoma of the larynx.

**Œsophageal.** Internal: Ingested foreign body such as a bone. Intrinsic: Œsophagitis; ulcer; stricture; spasm; globus hystericus; tumours; diverticula; achalasia of the cardia; paralysis (bilateral lesions of the vagus). External: Enlarged cervical glands; tumours of the thyroid; mediastinal tumours; aneurysm of the aorta; dilatation of the left auricle; dissecting aneurysm; congenital abnormalities such as right-sided aortic arch, double aortic arch and aberrant right subclavian artery; pleural or pericardial effusion; unilateral pulmonary fibrosis.

## THE STOMACH

**Introductory.** In the majority of cases of disorders of the stomach the physical signs are slight and inconclusive, and diagnosis depends on a very careful investigation of the history and symptoms, and on special investigations by test meals, opaque meals and gastroscopy.

### Gastritis

**Definition.** Inflammation of the mucous membrane of the stomach. This may be acute or chronic.

### Acute Gastritis

**Etiology.** Acute gastritis usually results from the ingestion of some irritant such as articles of food either indigestible, unmasticated or tainted (food poisoning), from excessive amounts of alcohol or from poisons such as perchloride of mercury, etc. During the 1914-18 war it was found associated with mustard gas and blue-cross gas poisoning. In children, it forms a part of summer diarrhoea (acute gastro-enteritis). Acute gastritis may occur in influenza, uræmia, pneumonia, bronchitis

or rarely in typhoid or typhus fevers. The infection here is probably hæmatogenous. In pyæmia and small pox suppurative gastritis may occur.

**Pathology** The mucous membrane of the stomach is red and inflamed and actual hæmorrhages may occur. In infective cases there may be local or diffuse suppuration in the submucous tissues (phlegmonous gastritis) and this may cause perforation of the stomach wall.

**Clinical Findings** The patient is usually an adult who gives a history of any of the causative conditions enumerated above. He complains of being suddenly taken ill with pain in the epigastrium followed often by vomiting and thirst. The vomit at first consists of stomach contents, but later there is usually little beyond mucus gastric juice and possibly blood. In severe cases there are general toxic symptoms with prostration, faintness, pallor, subnormal temperature and a rapid feeble pulse. The temperature may be raised in cases due to food poisoning. If the irritant enters the intestine there may also be diarrhoea.

**Differential Diagnosis** The diagnosis is usually clear from the history and picture of the case. The vomit should be examined in order to determine if possible, the cause of the illness. The acute abdominal pain may suggest abdominal angina or a tabetic crisis.

**Course and Complications** In the majority of cases the illness is of short duration, and complications apart from diarrhoea, are rare. In severe cases the sequelæ include ulceration of the stomach, chronic gastritis and gastric stenosis.

**Prognosis** This depends on the nature of the irritant. Death is inevitable with phlegmonous gastritis and may occur rapidly in poisoning cases.

**Treatment** If poisoning is suspected a specimen of the vomit should be saved in a clean jar for special tests to determine the nature of the irritant. The patient should be put to bed and only sips of water allowed by mouth. The bed should be warmed with hot bottles. In cases of poisoning not due to a corrosive substance a stomach tube should be passed and the stomach washed out. In poisoning due to corrosives the appropriate antidote should be given. Subsequently only small quantities of water are allowed by mouth (see p. 73).

In cases not due to poisoning a preliminary dose of castor oil  $\text{fl oz } \frac{1}{2}$  should be given followed by a gastric sedative, such as Sod bicarb  $\text{gr } 10$ , bism carb  $\text{gr } 15$ , acid hydrocyan dil  $\text{m } 2$ , muc acac  $\text{fl oz } \frac{1}{2}$ , aq ad  $\text{fl oz } 1$ . Fl oz 1 every 4 hours.

To relieve epigastric pain, hot flannels or a mustard leaf may be applied. If there has been much loss of fluid by vomiting rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours. In cases of severe collapse a stimulant such as Coramine (nikethamidum B.P. Add.) 15 mil should be injected hypodermically, and 4 fl oz of strong hot coffee may be administered per rectum.

Food should be given with caution when vomiting and acute pain

have ceased. Three ounce feeds of citrated milk (gr. 2 to fl. oz. 1) or arrowroot every 2 hours are most suitable.

### Chronic Gastritis

**Etiology.** The most common primary causes are chronic over-indulgence in alcohol, tea rich in tannin, over-eating, piquant foods such as curries, cold foods such as ices, excessive smoking, and improper mastication of food. Chronic gastritis may be secondary to acute gastritis, to local causes such as an ulcer or growth in the stomach, or to passive venous congestion in association with cirrhosis of the liver and heart failure. Chronic gastritis may also occur in wasting diseases such as carcinoma or tuberculosis, or in blood diseases such as pernicious anæmia or leukæmia.

**Pathology.** There is excessive secretion of mucus with later some atrophy of the mucous membrane. Venous congestion is noticeable in cases due to back pressure.

**Microscopically.** in early cases there is round-celled infiltration between the glands; later the glands may disappear, and the mucous membrane be converted into granulation tissue. In some instances the gastritis is localised to the pyloric region and erosions may be found.

**Clinical Findings.** The patient is usually an adult who complains of "indigestion." The chief symptoms are a poor appetite, an unpleasant taste in the mouth, regurgitation of food, heartburn or excessive thirst. The bowels may be costive or loose; in the latter instance there is usually deficient gastric acid secretion. Flatulence and heartburn may result from fermentation processes. In chronic alcoholism there is nausea and morning vomiting, chiefly of mucus or glairy fluid. Hæmatemesis may occur in gastritis due to venous stasis.

**On Examination:** The tongue is coated, white or brown, the complexion is usually sallow, but telangiectases may be seen on the face and dilated venules in the conjunctivæ in cases of cirrhosis of the liver. There are no definite abdominal signs, but diffuse tenderness may be elicited in the epigastrium. A fractional test meal shows excess of mucus in the majority of the specimens and the free acid is usually low or may be absent throughout. Fermentation acids are often present. The opaque meal in primary cases shows no evidence of ulcer or growth. Gastroscopy is of most value in the diagnosis of gastritis. During the active stages the mucous membrane shows hyperæmia, œdema, exudation, hæmorrhages and superficial ulceration. The results of the inflammation are granularity of the mucous membrane, narrowing, distortion and atrophy of the folds. In juxta-pyloric gastritis the clinical findings simulate those of duodenal ulcer, e.g. hunger pain relieved by food, gastric hypersecretion and small hæmorrhages due to erosions. In such cases the occult blood test in the fæces is positive.

**Differential Diagnosis.** Other causes of gastric pain must be excluded. The diagnosis is established by the history and symptoms of the case, the test meal and opaque meal findings.

**Course and Complications** Chronic gastritis is often a prolonged affection, unless the primary cause can be removed.

**Prognosis.** This is largely dependent on that of the primary condition.

**Treatment** The patient should be strictly dieted, irritant substances such as curries, mustard, stewed tea, greasy foods and heavy meals being avoided, and no meat must be eaten for some weeks. Meals must be taken at regular intervals. Alcohol and smoking should be forbidden or strictly limited. Washing out the stomach with a pint of warm sodium bicarbonate solution (gr 60 to fl oz 20) or with hydrogen peroxide (10 vols) in warm water (m 30 to fl oz 20), through a wide bore stomach tube helps to get rid of excess of mucus. This can be done night and morning for a few days in obstinate cases, and subsequently a glass of hot water should be drunk before meals. Oral sepsis should be eliminated and adequate mastication enforced. An alkaline gentian mixture with nuxvomica helps to stimulate the appetite, such as Tnc nuc.vom m 10, sod bicarb gr 15, sp.chlorof m 7, infus gent co. rec ad fl oz 1. Fl oz 1 t d s a c. If there is deficient free hydrochloric acid this should be supplied as acid hydrochlor dil m 30 to 60 in 6 fl oz of water with syrup auranti m 30 and some sugar to taste, 3 times daily with and after meals. Diarrhoea is usually checked by the acid prescription, whereas constipation can be overcome by adding liquid extract of cascara m 10 to 20 to the alkaline gentian mixture, or by giving liquid paraffin fl oz  $\frac{1}{2}$  to fl oz 1 at night. In cases with hypersecretion the treatment is as for gastric or duodenal ulcer (see p. 36).

### The Gastric Dyspepsias

**Definition** Disturbances of stomach function.

**Etiology.** The main function of the stomach is to liquefy food and pass it on to the duodenum. Disturbances of function may be secretory, muscular or nervous. The following clinical varieties will be described. Hyposecretion, including achylia. Hypersecretion, including Reichmann's disease. Gastric flatulence, including aerophagy. Gastroparesis (see p. 64). Bulimia. Anorexia and anorexia nervosa. Heartburn and waterbrash.

#### Hyposecretion

(Including Hypochlorhydria, Achlorhydria and Achylia Gastrica)

**Definition** Diminished secretion of gastric juice. The term is usually employed to mean diminished secretion of hydrochloric acid.

In achlorhydria there is absence of free hydrochloric acid and in achylia gastrica there is a complete absence of free hydrochloric acid and pepsin in the gastric juice.

**Etiology** Hypochlorhydria may be met with in apparently healthy individuals, or may result from emotion, worry, fatigue, or chronic gastritis. Achlorhydria may also occur in apparently healthy individuals or may result from chronic gastritis or carcinoma of the stomach. It may also occur with simple achlorhydric anaemia (see p. 422), acne rosacea, rheumatoid arthritis, chronic appendicitis or



*cholecystitis*. Some authors believe the achlorhydria is always caused by gastritis which may be due to hæmatogenous toxins resulting from infections either before or after birth. Achlorhydria is more common in women. It is rare with duodenal ulcer, less uncommon with gastric ulcer. It occurs with such allergic states as asthma, migraine or urticaria. Achylia gastrica is usually present with pernicious anæmia.

**Clinical Findings.** Frequently there are no symptoms. In other cases the patient, usually an adult, complains of abdominal discomfort and fulness after a small meal, flatulence, anorexia, heartburn and diarrhœa. A test meal shows a complete absence of free hydrochloric acid and a low total acid curve. The opaque meal shows rapid stomach emptying due to absence of acid in the duodenum. Normally acid in the duodenum causes the pylorus to close until it is neutralised by the pancreatic secretion.

**Treatment.** Dilute hydrochloric acid should be given by mouth immediately after or with the later stages of the meals. Doses of m. 5 to m. 60 may be required, flavoured with sugar or syrup of orange in a glass of water. In some cases the acid causes irritation of the bladder and frequency of micturition. *Tuc. hyoseyam. m. 30* and *pot. cit. gr. 15* can then be given at night.

### Hypersecretion and Hyperchlorhydria

**Definition.** Hypersecretion (Reichmann's disease) is an excessive secretion of gastric juice. The hydrochloric acid is not usually increased in strength in the secretion, although the total volume of hydrochloric acid excreted is necessarily increased. In hyperchlorhydria the volume of gastric juice may not be increased, but the concentration of hydrochloric acid is greater than the normal of about 0.1 to 0.2%, as obtained with the test meal.

**Etiology.** Hypersecretion may occur in apparently normal individuals, in nervous states, or in association with delayed stomach emptying. This may be due to reflex spasm of the pylorus and gastric ulceration.

Hyperchlorhydria occurs in about 5% of apparently normal men, or in association with pylorospasm and juxta-pyloric ulcer, the gastric crises of tabes, and at times in association with chronic cholecystitis or appendicitis.

**Clinical Findings.** The patient with hypersecretion may be in perfect health, or complain of vague symptoms of abdominal discomfort after meals, or of pain about 3 hours after food. A stomach splash is elicited over 4 hours after a meal. The typical fractional test meal findings are an excessive fasting stomach content (over 50 c.c.) and an excessive residue, such as 400 c.c. of clear gastric juice 3 hours after the meal has been given. With the barium meal the excessive gastric secretion may be seen in the stomach above the barium. In physiological hyperchlorhydria there are no symptoms. A test meal shows usually a high climbing acid curve, but there is no excess of fasting stomach contents, and no excess of residue at 3 hours.

**Treatment:** This is as for gastric ulcer (see p. 36).

## Gastric Flatulence and Aerophagy

*(Flatulent Dyspepsia)*

**Definition.** Distention of the stomach with gas

**Etiology** The modern view is to regard flatulence as being due to air swallowing (aerophagy) There appears however, little doubt that clinically in many cases it is due to fermentative changes in the stomach, usually associated with gastric hyposecretion More rarely it is caused by aerophagy (a neurosis) or perhaps by deficient absorption of air from the stomach owing to venous stasis from heart failure or cirrhosis of the liver.

**Clinical Findings** The patient complains of epigastric distention after meals, fulness and eructations of wind Flatulent dyspepsia is a prominent symptom in many cases of gall bladder disease It often gives rise to palpitations and pain near the apex of the heart, which is mistaken by the patient for heart disease As a psychoneurosis, aerophagy becomes a morbid habit, the patient constantly swallowing and belching up wind, with noises which are very distressing for those near him Aerophagy may also occur with acute dilatation of the stomach (see p 28)

**Treatment** In flatulent dyspepsia the meals should be given dry, and fluid drunk half an hour before, or 2 hours after meals A carminative such as ol cajaput m 2 or ol terebin m 10 may be given on sugar, or Sod bicarb gr 10, sp ammon aromat m 20 sp chlorof m 5, infus caryophyll rec ad fl oz 1 Fl oz 1 three times daily after meals Taka diastase may be given in an alkaline mixture such as Sod bicarb gr 10, Taka Diastase Liq m 80, sp chlorof m 7, infus gent co rec ad fl oz 1 Fl oz 1 t d s p c

In aerophagy due to nervous causes the nature of the air swallowing should be explained, and the patient instructed to resist all desire to "bring up" the wind He will then be unable to swallow more. In some cases the suggestive effect of passing a stomach tube results in a cure

**Bulimia**

A condition of excessive hunger which may be met with in diabetes mellitus, gastric ulcer, hyperthyroidism, or as a neurosis

**Anorexia Nervosa***(Hysterical Anorexia)*

A condition characterised by complete loss of appetite without any organic cause being discoverable The loss of appetite was attributed by Sir W M Gull to a "morbid mental state," and it is thought by some that this leads to a functional disturbance of the anterior lobe of the pituitary The condition in some ways resembles Simmonds disease (see p 672) The latter, however usually occurs at a later age period Predisposing causes include emotional crises, unhappiness at home, at school or at work "slimming," an operation or a severe illness The patient is usually a young woman aged 15 to 20 years, who refuses to

take any food, steadily loses weight and suffers from amenorrhœa. The amenorrhœa may begin before, or at the same time as the anorexia. There is an increased downy growth of hair on the trunk, limbs, lips and chin, and the pubic hair may show the male distribution. She is active and restless and displays complete indifference towards her symptoms. Death is liable to occur from what is apparently a functional disease.

The nature of the illness should be explained to the patient and her relatives. She should be isolated and small feeds given two hourly, the nurse or doctor ensuring that each feed is eaten. If necessary the patient should be fed through a stomach tube at the commencement. The administration of thyroideum gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  daily may help to stimulate the appetite. The patient cannot be considered to be cured until her monthly periods are restored.

### Heartburn

Heartburn is a burning sensation felt behind the sternum or in the epigastrium, usually accompanied by regurgitation of a little acid fluid into the mouth. It may occur either with hypersecretion, due to hydrochloric acid, or with hypochlorhydria, due to fermentation acids. In the former case alkalis are required after meals, in the latter dilute hydrochloric acid.

### Waterbrash

Waterbrash is a regurgitation of acid fluid into the œsophagus accompanied by a copious secretion of saliva. The latter may run out of the mouth and be followed by vomiting. It is usually associated with hypersecretion and relieved by alkalis.

### Dilatation of the Stomach

#### (Gastrectasis)

There are two types, acute and chronic.

#### Acute Dilatation of the Stomach

##### (Acute Paralysis of the Stomach)

**Etiology.** The dilatation may be due to paralysis of the gastric branches of the vagus or to overactivity of the sympathetic, associated with a kink of the third part of the duodenum, where it is crossed by the superior mesenteric vessels. This kink is produced by the drag of the dilated stomach. Some writers believe that the dilatation is initiated by air swallowing. It usually follows operations such as prostatectomy, but it may occur in any severe illness, after childbirth, after injuries to the spine, femur or head, or following a heavy meal.

**Clinical Findings.** Shortly after coming round from the anæsthetic after an abdominal operation the patient notices pain or oppression in the epigastrium, and later large quantities of watery fluid containing mucus or bile pour out from the mouth.

**On Examination:** In the early stages there is slight fulness in both hypochondria. Later the patient is pale and collapsed, the abdomen is distended and sometimes tender; a stomach splash is elicited. The

pulse is rapid and of poor volume, and the temperature is usually subnormal

**Differential Diagnosis** Acute dilatation of the stomach must be differentiated from peritonitis or intestinal obstruction. The absence of fever and fecal vomiting, together with the signs of a grossly dilated stomach, are characteristic findings

**Course and Complications** If untreated it may prove rapidly progressive

**Prognosis** This is serious, and death may rapidly occur unless treatment proves successful

**Treatment Prophylactic** This consists in the avoidance of tight abdominal binders after operations. Mouth washes should be used to prevent air swallowing

**Curative** If the condition is diagnosed sufficiently early and adequate treatment applied it is not usually necessary to place the patient in the knee elbow position or on his face. As soon as it is suspected a Ryle's tube should be passed through the nose, and the stomach contents aspirated with a well fitting glass syringe. If excessive fluid is obtained, indicating dilatation the stomach should be kept empty by a continuous suction apparatus attached to the Ryle's tube. Normal saline should be given intravenously by the continuous drip method to replace lost fluid, and the patient should be allowed fluid by mouth as desired. Physostigmine salicylate gr 1/200 should be given subcutaneously every 4 hours for 3 doses

### Chronic Dilatation of the Stomach

This may be non-obstructive or obstructive

#### Non-obstructive Dilatation of the Stomach

**Etiology** The dilatation may result from atony of the stomach, due to chronic gastritis, over eating or over-drinking, or be associated with general weakness, visceroptosis or convalescence from severe illnesses such as enteric fever

**Pathology** The stomach is dilated and holds over 2 pints, but very much larger quantities have been recorded. The muscle wall becomes thin and the mucous membrane chronically inflamed or atrophied. Gaseous fermentation helps to distend the stomach. Generalised visceroptosis is often present

**Clinical Findings** The patient is usually a woman, who complains of digestive disturbances especially epigastric fulness and distention after meals with eructation of food and occasional vomiting, sometimes of large quantities of food. She usually suffers from chronic constipation

**On Examination** The general nutrition is often poor, the condition of the abdominal wall suggests visceroptosis (see p 64) and a marked stomach splash is found lasting over 4 hours after a meal. The fractional test meal shows a delay in stomach emptying, excess of mucus in the specimens and usually a diminution in the free hydrochloric acid content. An x-ray examination with a barium meal shows

the abnormal size and position of the stomach and the rate of emptying.

**Treatment.** The patient should take meals dry, and have smaller meals than usual at more frequent intervals, such as 6 times a day. At least 8 pints of fluid should be drunk in the 24 hours, but apart from meals. Constipation should be corrected by drugs such as ext. cascarr. sagrad. sicc. gr. 2 to 4, paraffin. liq. fl. oz.  $\frac{1}{2}$  to 1, or confection. sennæ gr. 60 nocte. Abdominal exercises as for visceroptosis (see p. 66) and the wearing of an abdominal belt are often helpful. The patient should masticate well, eat slowly, and lie down for 20 minutes on the right side after meals. Milk puddings, soups and greasy foods should be avoided. A digestive tonic is helpful, such as Tnc. nuc. vom. m. 7, glycerin. pepsin. m. 60, Taka-Diastase Liq. m. 60, sp. chlorof. m. 7, infus. gent. co. rec. ad fl. oz. 1. Fl. oz. 1 t.d.s. p.c. Alternatively, a course of arsenic may be given using sod. cacodyl. gr. 1 daily, injected intramuscularly 6 days a week. The total amount injected in a course is 20 grains.

### Obstructive Dilatation of the Stomach

**Etiology.** Obstructive dilatation may result from pyloric obstruction, due to an ulcer, new growth, congenital stenosis, pylorospasm or external adhesions, or to an hour-glass stomach.

**Pathology.** The stomach is usually considerably enlarged and some hypertrophy of its musculature may occur.

**Clinical Findings.** The patient is often an adult male, who complains of indigestion associated with gradual weakness. There is epigastric discomfort, flatulent distention and periodical vomiting of large quantities of sour fluid, which has an offensive odour in malignant cases. The vomit often contains articles of food eaten several days previously. The appetite may remain good but constipation is severe.

**On Examination:** A marked stomach splash may be elicited several hours after the last meal and in some instances pyloric thickening is felt. Visible peristalsis, in which the waves pass from left to right in the upper abdomen, may be noted after abdominal palpation, and the outline of the distended stomach may be seen through a thin abdominal wall. Occasionally antiperistaltic waves are seen. A test meal shows stagnation of the stomach contents, excess of mucus, fermentation acids and a low or absent free hydrochloric acid content. Sarcinæ, yeast cells or Oppler-Boas bacilli may be present. An opaque meal indicates the size of the stomach and delay in emptying. This may be repeated after a course of tnc. belladon. m. 15 t.d.s., which is given for 3 to 4 days, to see whether the obstruction is due to pylorospasm. Pylorospasm may be due to apprehension, and some radiologists believe that the relaxation of the spasm at the second examination is due to the relief of this apprehension rather than to the effect of the belladonna.

**Course and Complications.** Organic pyloric stenosis is progressive, and, if due to carcinoma, usually rapidly so. Tetany may occur as a complication.

**Differential Diagnosis.** Dilatation of the stomach can usually be distinguished from that of the colon by clinical examination, and, in

some cases of the latter, removal of flatus by a rectal tube establishes the diagnosis. If the stomach is grossly dilated and filled with fluid, ascites or an ovarian cyst may have to be eliminated in making the diagnosis. There is usually no difficulty, as the dilated stomach can be emptied by a tube and the swelling thus removed. X rays also serve to establish the diagnosis.

**Prognosis** This depends on the cause of the obstruction and its amenability to treatment.

**Treatment** Gastro enterostomy is usually required in organic obstruction. Stenosis due to spasm may be relieved by daily gastric lavage and the administration of the belladonna 15 to 30 s.

### Congenital Hypertrophic Stenosis of the Pylorus

**Etiology** The stenosis is probably due to congenital hypertrophy of the circular fibres of the pylorus with superimposed pyloric spasm.

**Predisposing causes** 1 Age 2 to 4 weeks. 2 Sex Males predominate in the proportion of four to one. The first child of a family is especially prone to the affection. Phimosi is probably of no etiological significance.

**Pathology** There is pyloric thickening with hypertrophy of the circular muscle fibres, the stomach may be dilated. The duodenum is normal.

**Clinical Findings** The patient is usually a baby boy, either breast or bottle fed, aged about 2 to 4 weeks, who was healthy at birth but who has suffered from vomiting and constipation since he was about 2 weeks old. More rarely the vomiting has been present from birth. The vomiting is forcible or projectile, the fluid being ejected 2 to 3 feet and the baby loses weight. The vomit does not contain bile.

**On Examination** The baby is often pale, a little cyanosed and wasted. Visible peristalsis, from left to right, may be seen in the epigastrium after a feed. A tumour can be felt in nearly every case if sufficient care is taken. The warmed left hand is laid on the abdomen and the lower border of the liver defined. Pressure is then made with the fingers in the region of the pylorus and after the baby is fed the small tumour due to the thickened pylorus can usually be felt. It may be under the liver and only felt on inspiration and it may quickly relax. An X ray photograph may be taken, giving an ounce of milk and water and gr 60 of barium sulphate by a spoon, the delay in stomach emptying is thus revealed. Gastric analysis usually shows a high free and total acidity and absence of duodenal regurgitation. The vomiting leads to alkalosis, with a raised plasma bicarbonate and a lowered plasma chloride figure.

**Course and Complications** If untreated, the baby usually dies in a few weeks from starvation. Recovery may take 1 to 3 months with medical treatment. Gastro-enteritis due to cross infection in a hospital ward is a serious complication.

**Differential Diagnosis.** The condition must be diagnosed from other causes of vomiting. The characteristic features are the age of onset, the projectile vomiting, visible peristalsis and most important

the palpable tumour. In congenital duodenal stenosis the vomiting begins at birth and bile is present in the vomit.

**Prognosis.** A certain proportion of patients recover spontaneously, but as it is impossible to say whether an individual one is going to do so, treatment should be given immediately the diagnosis is made. There is a difference of opinion as to whether this treatment should be medical or surgical, and whether surgical treatment should only be given if medical fails. On statistical evidence the palm must be awarded to the surgeons, as in a series of 50 cases operated on in nursing homes 100% recovered, and with a series of 100 cases, who were *breast-fed* before and after the operation, and were operated on in hospital, all recovered. On the other hand, with bottle-fed infants operated on in hospital, a mortality of 10% must be expected, death being usually due to gastro-enteritis acquired by cross-infection in hospital.

**Treatment. Surgical.** The operation is that of Rammstedt's pyloromyotomy, the pylorus being divided longitudinally down to the mucous membrane. It is usually performed under local anaesthesia, the stomach being washed out immediately before the operation.

**Medical.** This consisted formerly of gastric lavage, using a No. 6 or 7 Jacques soft rubber catheter and a 1% sodium chloride solution at 100° F., twice daily just before a feed. Small hourly feeds were given of 1 to 2 teaspoonfuls of breast milk which had just been drawn off, or of citrated or peptonised milk. The feeds were gradually increased to 1½ oz. The best medical results are obtained by the use of Eumydrin (atropine methylmtras), the mortality in Gothenburg being 1%, whereas in England with hospital cases the mortality is about 11.5%, between 70% and 80% being cured. Eumydrin may be given by mouth using a 1 in 10,000 solution in water, the first dose is 0.5 to 1 mil., increasing by 0.5 mil. at each feed until 2 to 3 mils are given six times daily. The Eumydrin is administered half an hour before the feeds, which are given three hourly. The treatment has to be continued for about 4 weeks. Eumydrin (0.6% in alcohol) may also be given by drops applied to the tongue. One drop twice a day may prove sufficient to stop the vomiting. It is a mistake to administer too much fluid by mouth, or in the form of normal saline subcutaneously, unless the patient is very dehydrated, as this diminishes the effect of the drug. The total fluid required (between feeds) is 3 oz. per lb. body weight during the first 24 hours, subsequently the infant is given by mouth between feeds as much fluid as he requires in addition to his feeds. Gastric lavage is not necessary. Toxic effects include rise of temperature, flushing of the skin and abdominal distension. These can usually be relieved by omitting the next dose of Eumydrin. The duration of the treatment is likely to be 28 days in hospital, during which time there is a grave risk of gastro-enteritis occurring from cross-infection unless adequate precautions are taken as regards isolation and nursing.

### Hypertrophic Stenosis of the Pylorus in Adults

A few cases of hypertrophic stenosis of the pylorus of the congenital type have been met with in adults. They are characterised by recur-

rent attacks of vomiting which may be associated with tetany, without necessarily a history of infantile vomiting. The condition can be cured by pyloroplasty, if feasible, or relieved by gastro-enterostomy.

### Hæmatemesis

**Definition** Vomiting of blood

**Etiology.** The blood may be derived from various sites —

*The Stomach (gastro-rhagia)*

**Local causes** Erosion (gastrostaxis, bleeding from minute foci), ulcer, carcinoma or sarcoma, acute gastritis, trauma, corrosives, simple tumours such as polypi or angiomas, a tuberculoma, a gumma. Intra-gastric rupture of aneurysm of the aorta. *Tabes dorsalis* (gastric crises)

Portal congestion due to cirrhosis hepatis, splenic anæmia, heart failure, thrombosis of the portal vein

**Toxic and infective causes** Yellow fever, Weil's disease, small pox, hæmorrhagic scarlet fever, hæmorrhagic measles, appendicitis, cholecystitis, septicæmia, influenza, cholæmia, uræmia

**Blood diseases** Leukæmia, purpura, pernicious anæmia, hæmophilia, erythæmia

*The Œsophagus* Ruptured varicose veins in cirrhosis of the liver

*The Duodenum* An ulcer

*The Lungs, Nose or Mouth* The blood from these sources is swallowed before it is vomited

**Clinical Findings** If the hæmatemesis is severe, the patient usually experiences a preliminary feeling of faintness with nausea, and then vomits up the blood

**On Examination** The patient is often pale and blanched. The vomited blood tends to be dark (coffee grounds) and is acid in reaction unless a large dose of alkali has been taken just before the vomiting. Food may be present in the vomit and the blood is not aerated, as in hæmoptysis. The stools subsequently are dark and tarry (melæna) for about two days after the bleeding has ceased

**Treatment** This depends on the cause. The routine treatment for hæmorrhage due to gastric ulceration is described on p. 39

### Vomiting

**Definition** Expulsion of the stomach contents from the mouth by abdominal and diaphragmatic contractions

**Etiology** Vomiting may be due to — *Gastric Causes* Dyspepsia, gastritis, ulcer, carcinoma, congestion in heart failure or cirrhosis, hour glass constriction, pyloric obstruction, emetics such as salt or mustard, irritants such as arsenic, digitalis, poison gases, etc

*Central Causes* Stimulation of the vomiting centre in the medulla by anæsthetics, apomorphine, tobacco, toxins in uræmia, alkalosis, acidosis in diabetes mellitus and cyclical vomiting, cholæmia, pregnancy, Graves' disease, Addison's disease, scarlet fever, influenza, acute yellow atrophy of the liver

*Nervous Causes* Emotions, hysteria, migraine, concussion,



meningitis; intracranial tumours, abscess or hæmorrhage; tabes dorsalis.

*Reflex Causes:* Pharyngeal irritation; intestinal obstruction; appendicitis; worms; peritonitis; acute pancreatitis; biliary colic; renal colic; Dietl's crisis; vestibular causes as in Ménière's disease and possibly seasickness; nasal causes, due to odours; uterine and ovarian irritation, as in pregnancy; testicular trauma.

### Gastric Ulcer

#### (*Peptic Ulcer*)

**Definition.** A simple ulcer of the stomach of doubtful etiology.

**Etiology.** The cause is unknown. Gastric juice appears to be an essential factor in the ulcer production, as peptic ulcer occurs only in situations exposed to its action, such as the stomach, first part of the duodenum, last part of the œsophagus, and in the jejunum after gastric anastomosis (anastomotic ulcer). A peptic ulcer may develop in an area of heterotropic gastric mucous membrane in a Meckel's diverticulum. Other factors which may play a part in its production are: Trauma from ingested irritant food. Focal sepsis as in the mouth, appendix or gall-bladder, with local action of toxins or microbic emboli in the gastric mucosa. Arterial spasm in the mucous membrane of the stomach. Arterial thrombosis. Nervous disturbances, such as overactivity of the vagus or underactivity of the sympathetic, which may result from tobacco. Worry appears to predispose to hæmorrhage and perforation. Stimulation of a vegetative centre in the inter-brain by Pituitrin or by a tumour, with consequent increased vagal activity, may be a factor in some cases. The posterior lobe of the pituitary has been shown in animals to produce a gastrototoxic substance which can cause lesions in the acid-bearing area of the stomach. There may also be a gastric ulcer diathesis, as gastric ulcers tend to run in some families and duodenal ulcers in others.

**Pathology.** The ulcer varies in size from that of a silver threepenny piece to that of a florin or even larger; it is usually about the size of a shilling and is commonly circular or oval. Multiple ulcers may occur. Acute and chronic ulcers are described. The former are usually small and often multiple, their floor is smooth and adhesions are not found. In chronic ulcers there may be much fibrosis producing pyloric stenosis or hour-glass constriction of the stomach. The following are the most frequent sites: Near the pylorus, on the lesser curvature, on the posterior wall, on the anterior wall and cardia, on the greater curvature. The ulcer is sharply delineated and extends a variable distance through the stomach wall. It may erode and perforate the stomach wall, or be surrounded by inflammatory tissue, or become adherent to such organs as the pancreas.

Other changes found in association with gastric ulcer are perforation of an artery in the ulcer, perigastric suppuration due to localised peritonitis which may cause a subphrenic abscess, generalised peritonitis from perforation, fistulæ with the colon, duodenum or pleura, and peri-

gastric adhesions to the liver, gall bladder, etc. Carcinoma rarely develops in an old gastric ulcer, but healing with a resultant scar is not infrequent.

**Clinical Findings** The patient who has an acute ulcer is often an anæmic girl, the first symptom being hæmatemesis. Chronic ulcers are equally common in males or females. The patient is usually an adult, over the age of 20, although a gastric ulcer has been recorded in a child aged three months. There is a history of periodic attacks of indigestion. The characteristic features of the attack are epigastric pain appearing a quarter of an hour to one hour after food and disappearing before the next meal. The pain is believed to be caused by muscular contractions or spasm of the stomach. It may be felt in the epigastrium to the left of the mid line or near the xiphisternum and may pass through to the back near the angle of the left scapula. It is often relieved by vomiting or by alkali medicines. Other symptoms such as nausea, vomiting, hæmatemesis or melæna may occur. In some instances, especially when the ulcer is on the lesser curvature of the stomach or near the cardiac end, there are no symptoms until a severe hæmatemesis occurs. When the ulcer is situated close to the pylorus the symptoms resemble those of a duodenal ulcer. The appetite is often good, but the patient is afraid to satisfy it. Other symptoms such as heartburn or flatulence with epigastric distention may be troublesome.

**On Examination** The patient is often well nourished, but the tongue is usually furred and oral sepsis is frequently present. Tenderness and cutaneous hyperalgesia may be found in the epigastrium, the tender spot corresponding with the site of the pain as demonstrated by the patient with his finger tip. Some muscular rigidity of the upper rectus muscle may be felt on one or both sides. An opaque meal may demonstrate the ulcer as a projecting mass of barium in the lesser curvature, in other cases a notch or permanent filling defect may be seen in the stomach wall. Pyloric spasm may be present with juxta pyloric ulceration. An incisura or hour glass constriction may be noted in some cases with mid gastric ulcers. If a constriction of the stomach wall is noticed, the opaque meal should be repeated after the patient has been given a course of belladonna, such as the belladon in 15, tds a c for 3 to 4 days, to see if the constriction is due to spasm or to organic deformity. A muscular spasm is often seen in the stomach wall opposite the site of the ulcer. A fractional test meal shows a climbing type of curve with high acidity if the ulcer is near the pylorus and pylorospasm is present. Blood may be found in the specimens removed. In at least 50% of cases the test meal findings are normal especially if the ulcer is not juxta pyloric. Achlorhydria practically never occurs with gastric ulcer. In some cases gastroscopy will reveal an ulcer which is not demonstrable radiologically, the converse is also true. A gastro-jejunal stoma is not always easy to see. The faeces. The occult blood test may be positive. For this purpose a specimen of the faeces should be examined after the patient has taken no meat, fish, gravy or green vegetables for 3 days. There are many atypical cases

in which the history and symptoms do not suggest gastric ulcer, and yet its presence is revealed by the opaque meal.

**Differential Diagnosis.** In very many cases a presumptive diagnosis of gastric ulcer can only be made after X-ray and test meal examinations, the symptoms and signs being entirely misleading. In other cases the diagnosis is established by gastroscopy or laparotomy. Such conditions as gastritis, carcinoma of the stomach, tabetic crises, chronic appendicitis, chronic cholecystitis and gastric neuroses may all cause symptoms closely resembling those of gastric ulcer. Carcinoma should be suspected if the pain is not relieved by a week's strict treatment.

**Course and Complications.** *Acute gastric ulcers:* These usually heal rapidly when the appropriate treatment is carried out. *Chronic gastric ulcers:* These pursue an intermittent course. They may heal spontaneously, or progress and cause such complications as hæmorrhage, perforation, localised or general peritonitis, gastric stenosis due to pyloric or hour-glass constriction, perigastric abscess or adhesions, or carcinoma. General complications are anæmia and malnutrition.

**Prognosis.** This is on the whole good. A gastric ulcer rarely proves fatal apart from such complications as perforation, hæmorrhage or malignant change. Efficient medical treatment is improving the ultimate prognosis.

**Treatment.** In the majority of cases the patient requires a preliminary 4 to 6 weeks' course of treatment in bed followed by a prolonged after-treatment. The teeth should be attended to after dental radiograms have been taken, any focus of sepsis being eradicated. A modified Sippy diet is advisable with a neutralising substance after feeds, and olive oil, belladonna or atropine before the feeds, as described later. The treatment recommended is as follows:—

**Healing Stage (Diet 1).** *Weeks 1 and 2.* Feeds of 5 oz. are given at 7 a.m., 9 a.m., 11 a.m., 1 p.m., 3 p.m., 5 p.m., 7 p.m., 9 p.m., 11 p.m., and once during the night, if awake. The feeds consist of milk (warm and containing gr. 3 sodium citrate to the ounce), Horlick's malted milk or Benger's food. At least 5 of the feeds should be of milk, which may be coloured with tea. Strained orange or tomato juice, 1 oz., should be given daily throughout the treatment to supply vitamin C.

*Week 3.* Add 1 raw egg to two of the milk feeds. A little thin bread and butter is given with one feed, and cream 1 oz. 3 times a day. In addition, the patient may be given once a day a little of one of the following: Sweet jelly, milk jelly, custard or junket. The feeds are still given two-hourly.

*Week 4.* The feeds are given every 2½ hours, from 7 a.m. to 10 p.m. They consist of 5 oz. citrated milk every other feed, with 1 egg in two of the feeds, the thin bread and butter and the cream, as in Week 3. The alternate feeds are composed of a similar quantity of potato soup, arrowroot, cornflour, or milk pudding (sago or tapioca), and once a day a little milk jelly, sweet jelly, custard or junket.

*Week 5.* The feeds are given every 2½ hours. The milk may now be reduced to 3 times a day, at 7 a.m., 5 p.m. and 10 p.m. Additions to

the diet are pounded fish 2 oz, lightly boiled egg, crisp toast or rusk (well chewed) with butter.

*Weeks 6 to 8* Feeds still every 2½ hours Add 2 to 4 oz pounded fish or minced meat daily

Immediately before 3 feeds ½ fl oz of olive oil is given, and directly before 8 other feeds Tnc belladon m 5 to 10, aq chlorof ad fl oz ½, or Atropin sulph gr 1/200 in m 60 water is given The alkali or neutralising substance used may be magnesium trisilicate, a teaspoonful after feeds, or a 10% aluminium hydroxide gel, Aludrox, m 60 in water, 6 times a day between feeds, or a powder consisting of Mag hydrox, cretæ and bism carb equal parts, one teaspoonful of the powder in a little water is taken 1 hour after 5 feeds during the day, and 2 teaspoonfuls the last thing at night During the third and fourth weeks the alkali is taken 3 times a day after feeds, and a double dose at night In the fifth to eighth weeks the alkali is taken twice a day after feeds and a double dose at night The constitution of the powder can be modified according to the effect produced on the bowels, the mag hydrox being increased if there is constipation, the creta and bism carb, if there is diarrhoea Bismuth carbonate is out of fashion now owing to its expense, to the slaty colour it gives to the faeces which interferes with the detection of blood and because it is said neither to relieve pain nor to neutralise acidity The mouth should be cleansed after each feed with sod bicarb gr 60 in 5 fl oz of water No smoking is allowed

When the occult blood test is negative and there is no pain, the patient is put on the following diet —

*Convalescent Stage (Diet 2)* During this period of about a month the patient is allowed up for gradually increasing periods, and additions are made to the dietary

7 30 a m, *Breakfast* One egg (lightly boiled), thin bread and butter or crisp toast and butter (to be well chewed), honey or apple jelly, sugar and milky tea

10 a m Milk 5 oz (containing gr 15 sodium citrate) or Horlick's malted milk or Benger's food

12 30 p m, *Lunch* Milk soup (potato or artichoke), fresh white fish (boiled or steamed), or rabbit, chicken or tender mutton, mashed potato. Custard or milk pudding or stewed apples (with no pips, skin or core), and the juice of an orange

3 p m Thin bread and butter, or biscuits and butter, and milky tea

5 30 p m Milk 5 oz (containing gr 15 sodium citrate) or Horlick's malted milk

8 p m, *Dinner* As for lunch

10 30 p m Milk 5 oz (containing gr 15 sodium citrate) or Benger's food

The alkaline powder should be taken, 1 teaspoonful after breakfast and 2 teaspoonfuls last thing at night, and the olive oil or belladonna before breakfast, lunch and dinner The meals should be small and eaten slowly After about a month the after treatment is commenced

*After-treatment (Diet 3)* 8 a m, *Breakfast* A selection from

porridge, made from very fine oatmeal; egg, boiled, poached or scrambled; haddock boiled in milk; toast, crisply made and well chewed; breakfast biscuits, honey, apple jelly, butter, sugar, cream, and milky tea or coffee.

11 a.m. A glass of milk, Ovaltine, Horlick's malted milk, egg and milk, or Benger's food and a biscuit.

1 p.m., *Lunch*. A selection from soup, made from milk and vegetables, such as potato or artichoke. Fresh white fish, boiled, grilled or steamed with white sauce, or fish soufflé. Meat, such as minced fresh beef, mutton or veal, grilled lamb cutlet, boiled or roast chicken, sweetbreads, boiled tripe or boiled ham. Vegetables, such as mashed potato, steamed or boiled green vegetables, passed through a sieve and served with butter. Sweets, such as custard, boiled or baked; junket, milk pudding very well cooked, cornstarch, stewed apples with no pips, core or skin, stewed plums with no skin, chocolate soufflé. Crisp toast, the juice of one or more oranges, cheese soufflé.

4 p.m. A cup of milky tea, thin bread and butter, spongecake, honey or jelly.

7 p.m., *Dinner*. As at lunch.

10 p.m. A feed as at 11 a.m.

The breakfast, lunch and dinner should be small meals, well chewed and eaten slowly. The patient should rest for half an hour after meals. No condiments or vinegar should be eaten, and nothing taken which cannot be reduced to a soft pulp in the mouth. If it is impossible to obtain the liquid feeds between all the meals, a biscuit or some chocolate should be eaten. A teaspoonful of the alkaline powder should be taken every night on retiring to bed.

The following articles of food are forbidden: Smoked salmon, tough meat, high game, sausages, curry, made-up dishes, fried foods, cheese (except cream cheese), meat extracts and meat soups, pickles, salads, uncooked vegetables, new or wholenmeal bread, buns, unripe or raw fruit, nuts, raisins, jam with pips, marmalade with peel, strong tea or coffee, aerated drinks and alcohol.

The danger of alkalosis must always be borne in mind when the patient is taking large doses of certain alkalis. It is especially liable to occur with ulcers at or near the pylorus, when there is pylorospasm or renal insufficiency. The earliest symptom is often a distaste for milk. This is followed by headache, giddiness, nausea, vomiting, drowsiness, tetany or coma. The temperature is slightly raised, pulse frequent, and respirations slow. The blood nitrogen content rises, the blood pressure is often raised, and the alkaline urine may contain albumin and casts, the chloride excretion being low. All alkalis should be discontinued at once, and 1 oz. of dextrose given in water by mouth 3 times a day, or rectal injections of 4 to 8 fl. oz. of normal saline containing 5% dextrose should be given every 4 to 6 hours. Ammon. chlorid., 0.5 G. (stearetttes), should be given by mouth every 6 hours, or ammon. chlorid. 4 G. per rectum every 6 hours. An intravenous injection of 1 mil. of Collosoal Calcium should be given and repeated next day if necessary.

Any focus of sepsis in the nose, cranial sinuses, gall bladder or appendix should now be treated. The histidine treatment of gastric ulcer is not recommended.

**The Indications for Surgical Treatment** These are as follows: Perforation. Organic pyloric stenosis or hour glass constriction which impedes stomach emptying. Perigastric abscess. Intractable hæmorrhage due to a perforated, sclerosed artery, which will not repair by coagulation. A suspicion of carcinoma as aroused by the severity of the pain, loss of weight, the persistence of blood in the fæces, and the progressive fall in the free hydrochloric acid in the fractional test meal. Repeated hæmorrhages not responding to medical treatment or recurrent gastric ulcers may also require operation.

**The Treatment of Hæmatemesis** The patient should be put to bed and, providing the hæmatemesis is not due to cirrhosis of the liver, a subcutaneous injection of morphin sulph gr  $\frac{1}{4}$  and atropin sulph gr  $\frac{1}{100}$  given. This may be repeated up to a total of gr 1 morphine in the 24 hours. Nothing must be swallowed by mouth until the bleeding has stopped, as judged by the absence of hæmatemesis or melæna. The mouth may be washed out with a little cold water. An hourly pulse chart should be kept. The blood pressure and hæmoglobin percentage should be determined every 12 to 24 hours. The blood urea should be estimated, and, if raised, determinations should be made daily until it falls to normal. The bowels should not be opened for 4 to 7 days an enema then being given. Rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours, or a rectal drip may be used with a Murphy vulcanite rectal nozzle, which allows free escape of flatus. Six to 8 pints of normal saline (0.85% sodium chloride) and isotonic dextrose (5%) should be run in slowly every 24 hours, resting the bowel every other hour by interrupting the flow. Blood transfusion is required if the hæmoglobin is below 40%, the systolic blood pressure below 90 mm Hg, the pulse rate over 140, or the blood urea over 100 mg per 100 c.c. One pint of blood will increase the hæmoglobin by about 10%, and it should be raised to about 80%. By the drip method a 10% increase in hæmoglobin should be achieved every 4 hours. If the bleeding does not stop 5 mls of Coagulen Ciba may be injected intramuscularly twice a day, and a single injection of 10 mls of calcium gluconate (B.P. Add.) given. Five or ten mls of Stypven, containing 0.5 mg, or 1 mg of Russell's viper venom, given by mouth may arrest the bleeding. If there is a history of previous severe hæmorrhages, or if there is arteriosclerosis and the bleeding persists, an operation will probably be required. This should not be delayed until the patient is exsanguine, and a preliminary blood transfusion before the operation is of value. After 2 to 4 days, and when the bleeding has been arrested, as judged by absence of hæmatemesis and by the general condition of the patient, 4 fl oz of half strength normal saline (0.42% sodium chloride) should be given by mouth every 4 hours for 24 hours. Milk feeds are now begun: first milk 1 oz, water 1 oz and emuls mag oxid m 30 every 2 hours and then milk 2 oz every 2 hours, gradually increased to 5 oz every 2 hours.

The. belladon. m. 10 to 15 is given before 3 feeds and olive oil fl. oz.  $\frac{1}{2}$  alternately before 3 other feeds. A teaspoonful of the alkaline powder is given after 5 feeds, and a double dose at night. Ferri et ammon. cit. gr. 30 should be given t.i.d. between feeds. The patient should now begin the routine treatment for gastric ulcer. A barium meal should not be given within 2 months of a hæmatemesis as it may provoke a recurrence.

Meulengracht, in Copenhagen, has broken away from the traditional treatment of hæmatemesis. In a series of over 250 cases he has used a liberal dietary from the first day of the bleeding, and obtained the very low mortality of 1%. The treatment cannot be tolerated by severe cases of hæmatemesis. The diet is as follows:—

6 a.m. Tea, white bread and butter. 9 a.m. Oatmeal with milk, white bread and butter. 1 p.m. A selection from: Meat balls, timbale, broiled chops, fish balls, vegetable gratin, fish gratin, mashed potatoes, vegetable purée, vegetable soups, cream of vegetables, stewed apricots, apple sauce, gruel, rice and tapioca puddings. 3 p.m. Cocoa. 6 p.m. White bread and butter, sliced meats, cheese and tea. The patients are allowed to eat as much as they like. In addition, one teaspoonful of the following powder is given t.i.d.: Sod. bicarb. gr. 30, mag. carb. lev. gr. 80, ext. hyoscyam. sicc. gr. 4, and ferri lact. gr. 5, t.i.d.

### Carcinoma of the Stomach

**Etiology.** The cause is unknown. Most recent statistics tend to show that carcinoma rarely, if ever, develops from a chronic gastric ulcer. Chronic atrophic gastritis is an important predisposing condition. Carcinoma ventriculi is two to three times as common in the male as in the female sex. It is estimated that 4% of all adults die from it.

**Pathology.** The following types are described: 1. Scirrhus, a spheroidal celled carcinoma with much fibrous stroma. 2. Medullary or encephaloid, a spheroidal celled carcinoma with little fibrous stroma. 3. Adeno-carcinoma, a columnar celled growth. Dual (kiss) adeno-carcinoma has been described occurring on apposing surfaces of the stomach. In the so-called Krukenberg tumour secondary deposits occur in the ovary. 4. Leather-bottle stomach or *linitis plastica*, a diffuse scirrhus growth. 5. Colloid, the growth tends to spread to the omentum. 6. Malignant degeneration of a peptic ulcer. 7. Squamous celled carcinoma is rarely seen, and then it is at the cardiac end of the stomach.

The growth is most often situated at the pylorus, then on the lesser curvature, near the cardia, on the posterior wall, or diffusely on the anterior wall. Adeno-carcinoma may occur as a stomal growth, following a gastro-enterostomy. Secondary carcinoma of the stomach is rare, the primary lesion usually being in the breast.

**Clinical Findings.** The patient is often an adult male over the age of 40. He states that he has recently suffered from indigestion, a complaint formerly unknown to him or not noticed for several years. The earliest symptoms are usually abdominal discomfort or pain not definitely related to food and often present at night, followed by anorexia,

nausea and later pain aggravated by food, heartburn, eructations, flatulence, vomiting which may be very persistent regurgitation of mucus, general weakness, anæmia constipation, loss of weight and difficulty in digesting solid food. In other cases the early symptoms suggest a duodenal ulcer but without the typical periods of remission. Recurrent thromboses of veins in the legs may be an early symptom. Hæmatemesis is a rare occurrence but when present the vomit may show the typical coffee grounds appearance. The pain later becomes very severe.

*On Examination* In early cases nothing abnormal can be found beyond epigastric tenderness. As the disease progresses cachexia becomes noticeable, a palpable tumour due to the growth may be felt in the epigastrium or left hypochondrium, and enlarged glands may be felt above the left clavicle (Virchow's gland due to spread along the thoracic duct) in the left axilla, and in the groins. Nodules may be felt in the abdominal wall around the umbilicus. The liver may be enlarged and irregular due to secondary deposits, and ascites may result from pressure of enlarged glands in the portal fissure. In any case in which the suspicion of gastric carcinoma arises, the following investigations should be carried out.—*Opaque meal* This may show a large filling defect due to the growth protruding into the stomach and giving rise to gastric stenosis, or in early cases it is due to an interference with the normal peristaltic wave at a certain spot in the stomach as seen on the screen. *Fractional test meal* The typical findings are foul dark resting stomach contents with some blood and high fermentation acid but no free hydrochloric acid. Evidence of stagnation is shown by the presence of charcoal particles from the milk and charcoal feed given the night before. *Oppler Boas bacilli, sarcinæ and yeasts* Subsequent specimens show achlorhydria (absence of free hydrochloric acid) and the fermentation acids remain on the high side. Free hydrochloric acid may be found in 30% of cases. Achlorhydria is probably due to previous gastritis, and not to the growth involving the acid secreting area. The lactic acid in the stomach contents results from fermentation and is not a specific product of the growth. In some cases in which the X ray diagnosis of carcinoma is doubtful a definite diagnosis either positive or negative can be made by gastroscopy. *Occult blood in feces* A specimen of the feces should be tested for occult blood. If positive the test may be repeated after omitting all meat, fish, gravy and green vegetables from the dietary for 3 days. If still positive it indicates bleeding from the alimentary tract. The blood. There is usually severe anæmia which may approximate to the pernicious type, with some megalocytes and an occasional megaloblast, but the colour index is not usually above unity. There is usually a leucocytosis of 12 000 per c mm or over.

*Differential Diagnosis* The most important conditions to exclude are Dyspepsia from other causes, including ulcers of the stomach and duodenum. Authors of text books are often blamed for only giving a picture of advanced and inoperable carcinoma of the stomach. It should be suspected in all cases of digestive disturbances which do not quickly yield to adequate treatment. In all cases of doubt, full investi-



gations, as described above, should be carried out. Even then in some cases the diagnosis remains uncertain and can only be established by biopsy. Carcinoma of the stomach does at times occur in young adults, so it cannot be excluded on the ground of age alone. Pernicious anæmia: A blood examination should exclude pernicious anæmia, in which an achylia gastrica (see p. 489) is almost always present. Splenic anæmia: A swelling in the left hypochondrium associated with a hæmatemesis and anæmia may suggest an enlarged spleen, and splenic anæmia. The opaque meal usually serves to differentiate. A gumma of the stomach: The Wassermann reaction will help to exclude this, and if positive the response to vigorous anti-syphilitic treatment as described on p. 43 affords further confirmatory evidence.

**Course and Complications.** Death usually occurs within 6 to 12 months from the date of diagnosis, unless early surgical intervention proves successful. Complications include dilatation or rupture of the stomach, gastro-colic fistula, jaundice due to obstruction of the bile or hepatic ducts by secondary deposits, pleural effusion and thrombosis of the femoral vein.

**Prognosis.** This is usually hopeless unless early operation has been performed.

**Treatment.** If the investigations point to early carcinoma, laparotomy should be performed and the growth removed surgically if possible, or radium may be applied locally. Operation in the presence of clinical evidence of metastases, such as enlarged glands or liver, is not justifiable except to relieve pyloric obstruction. Deep X-ray treatment does not effect a cure.

**Medical treatment consists in:—Diet:** The patient should be given what he fancies. If there is gastric obstruction liquids or semi-solids only should be allowed. **Gastric lavage:** In cases of pyloric obstruction which are not operated on, the stomach should be washed out with sodium bicarbonate solution (gr. 60 in fl. oz. 20) daily or more frequently, using a rubber tube. **Drugs:** These are required for pain. *Nepenthe m. 20 to 30 t.i.d.* may be given by mouth, or *tnc. opii m. 30 t.i.d.* or *tnc. chlorof. et morphin. co. (B.P.C.) m. 5 t.i.d.* To secure sleep hypodermic injections of morphin. sulph. gr.  $\frac{1}{4}$  to 1 or *Omnopon (papaveretum B.P.C.) gr.  $\frac{1}{3}$*  are usually required. The anæmia usually improves temporarily with liver treatment (as for pernicious anæmia, see p. 490).

### Sarcoma of the Stomach

It is stated that about 1% of malignant growths in the stomach are sarcomatous. Growth is rapid. The age incidence is usually between 30 and 40. Clinically the condition can only be distinguished from carcinoma by operation. Secondary metastatic sarcoma may occur in the gastric wall.

### Benign Tumours, Cysts, and Foreign Bodies in the Stomach

These include: Polypus (adenomatous tumour), fibroma, myoma, adenomatous cyst, hydatid cyst and hair balls.

The treatment of these tumours is surgical.

### Tuberculosis of the Stomach

The stomach is very rarely affected by tuberculosis. An ulcer may occur secondary to pulmonary tuberculosis or miliary tubercles may be found in the mucous membrane.

### Syphilis of the Stomach

A gumma may cause pyloric obstruction, hour glass constriction or ulceration. Clinically it simulates peptic ulcer or carcinoma but the Wassermann reaction and the response to anti-syphilitic treatment establish the diagnosis. Some cases of *limitis plastica* are thought to be syphilitic in origin.

**Treatment** The patient should be given a course of iodides and mercury such as Pot iod gr 5 liq hydrarg perchlor m 20 sp chlorof m 7, aq ad fl oz 1. Fl oz 1 t d s p c. The iodide should be increased up to gr 30 to 60 t d s and be followed by a course of neoarsphenamine injections (see p 570).

## THE INTESTINES

### Duodenal Ulcer

**Definition** A peptic ulcer in the duodenum.

**Etiology** The underlying causes are the same as for gastric ulcer (see p 34). A duodenal diathesis is credited by some, characterised by a hypertonic, hypersecreting and rapidly emptying stomach. There is a distinct familial incidence. *Predisposing causes* 1 Sex. This disease is four times as common in men as in women. 2 Age. The usual age period is between 30 and 50 years. 3 Smoking. The effect of this is doubtful as in a series of over 400 cases over 80% were non smokers.

**Pathology** The ulcer is generally situated within an inch of the pylorus. Various stages may be found as with a gastric ulcer. The pain is considered to be due to pylorospasm.

**Clinical Findings** The patient is usually a middle aged male, who gives a history of discomfort, later succeeded by pain in the upper abdomen. The pain occurs periodically, lasting for a week or longer, with intervals of relief for several weeks or months. These relief periods shorten as the disease progresses. The pain is often localised by the finger to a spot in the epigastrium just to the right of the mid line. It may radiate through to the back near the inferior angle of the right scapula. The pain is related to food in that it occurs about 3 or 4 hours after a meal and tends to wake the patient at about 2 a.m. It is temporarily relieved by taking more food ('hunger pain') and by alkalis. The patient may say that an attack has followed some dietetic indiscretion. The appetite is usually good but the bowels are constipated. In some cases especially with an acute ulcer, melena, hæmatemesis or perforation may be the first indication of a duodenal ulcer.

**On Examination** The patient generally is not wasted. A tender spot may be found in the epigastrium in the subcostal plane, about

1½ inches to the right of the mid-line. The upper part of the right rectus muscle is then somewhat on guard. In order to diagnose a duodenal ulcer further tests are essential. They are:—An opaque meal: The stomach is usually of the hypertonic type and empties rapidly. Irregularity of the duodenal cap may be seen, or an ulcer crater in one wall, with a niche due to spasm opposite. If there is pyloric stenosis there is delay in stomach emptying. The fractional test meal: There may be no abnormality, but if the ulcer is at the "hurry" stage, there is a high climbing acid curve, due to pyloric spasm and rapid emptying due to violent gastric contractions. If there is pyloric stenosis the high acidity persists with delay in stomach emptying. The occult blood test: The faeces usually give a positive occult blood test, if active ulceration is present.

**Differential Diagnosis.** Many cases of duodenal ulcer are atypical. A gastric and duodenal ulcer may coexist, as demonstrated by X-rays. The symptoms may be those of gastric ulcer, whereas the X-ray examination indicates an ulcer in the duodenum. The duodenal symptoms may be due to duodenitis, or reflex, associated with chronic cholecystitis, appendicitis or renal lesions. The symptoms may be typical of cholecystitis, although the gall-bladder is normal and a duodenal ulcer is present. Duodenal adhesions may cause an X-ray deformity of the duodenal cap.

**Course and Complications.** There are various possibilities when a duodenal ulcer exists. They are:—Complete recovery without further symptoms, or in healing pyloric stenosis may develop with progressive vomiting. Progressive ulceration, with adhesions. Perforation: This may be a minute leak with very localised peritonitis, or a subphrenic abscess or generalised peritonitis may ensue. Haemorrhage. Chronic pancreatitis, with a rise of urinary diastase up to about 100 units, is met with in some cases. Duodenal ulcers do not become malignant.

**Prognosis.** This depends upon early recognition and adequate treatment. There is, however, a tendency to recurrence.

**Treatment.** This is identical with that detailed for gastric ulcer (see p. 86).

### Duodenal Obstruction

**Etiology. Acute Duodenal Obstruction.** This may occur as part of the mechanism resulting in acute dilatation of the stomach (see p. 28).

**Chronic Duodenal Obstruction.** This may result from:—

1. Progressive duodenal stenosis, associated with an ulcer.
2. External pressure, due to adhesions to the gall-bladder.
3. Visceroptosis, which may be confined to the duodenum.

The third part of the duodenum is constricted by the root of the mesentery and the superior mesenteric vessels; the first and second parts of the duodenum dilate, causing chronic duodenal ileus.

### Chronic Duodenal Ileus

**Clinical Findings.** The patient may experience no symptoms, the dilated duodenum being discovered on routine X-ray examination. In

other cases there are periodical attacks of illness, characterised by epigastric discomfort, gastric flatulence, which is succeeded by severe pain in some cases in the epigastrium to the right of the mid line, with vomiting of large quantities of bile stained fluid. In the intervals between the attacks the patient may feel perfectly well or suffer from malaise and headache. The diagnosis is established by a barium meal. Cholecystitis may occur as a complication.

**Treatment** Medical treatment consists in washing out the duodenum with a duodenal tube and in general treatment as for visceroptosis (see p 66). If this is not successful a posterior gastro-enterostomy or partial duodeno gastrectomy may afford relief.

### Duodenal Diverticula

These are occasionally met with. They often cause no symptoms and are discovered on X ray examination. In some cases persistent symptoms are present, such as epigastric discomfort 1 to 2 hours after food, flatulence, nausea and vomiting. The diverticula occur most often in the second part of the duodenum, on its concave side where the vessels enter and there is no peritoneal coat (see diverticulitis, p 16).

### Carcinoma of the Duodenum

This is a rare disease. It may start near the ampulla of Vater, or extend down the bile duct to the duodenum from a primary growth in the gall bladder, or invade the duodenum from a carcinoma of the head of the pancreas.

### Duodenitis

#### (*Proto duodenitis*)

**Definition** Inflammation of the supra papillary portion of the duodenum.

**Etiology** Duodenitis does not form a definite clinical syndrome, but it may occur as an early stage of a duodenal ulcer or of catarrhal jaundice. It may also be associated with a kink at the duodeno jejunal flexure.

**Clinical Findings** The patient may complain of pain in the epigastrium relieved by food.

**On Examination** Tenderness may be found over the duodenum.

**Treatment** This varies with the associated lesions.

### Jejunal and Gastro-Jejunal Ulcers

**Etiology** Jejunal or gastro-jejunal ulceration (anastomotic ulcer) occurs as a complication of gastro enterostomy performed for duodenal ulcer in about 1% to 2% of all such operations. It is very rare after a gastro enterostomy for gastric ulcer. It is met with therefore chiefly in males the important factor being the hyperchlorhydria. Other predisposing causes are persistent foci of infection in the nose and throat, cranial sinuses, gall bladder and appendix, and trauma due to pressure of the clamps at the operation or stitches which are not absorbed.

**Pathology.** The ulcer is usually single; it may occur at the site of anastomosis (gastro-jejunal) or in the efferent loop of the jejunum (jejunal), usually within an inch of the anastomosis.

**Clinical Findings.** The symptoms resemble those of duodenal ulcer. They may appear directly after the operation, or be delayed for as long as 17 years. The pain is usually located to a point just to the left of the umbilicus. Examination under X-rays with an opaque meal demonstrates a tender spot in the jejunum.

**Course and Complications.** Perforation may occur, with general peritonitis or the formation of a gastro-jejuno-colic fistula. Erosion of an artery may result in severe hæmorrhage.

**Prognosis.** Jejunal ulcers, if untreated, do not heal spontaneously. There is a tendency for recurrence after medical or surgical treatment.

**Treatment.** *Prophylactic.* Gastro-enterostomy should only be performed for duodenal ulcers causing obstruction. It is contra-indicated when the gastric acidity is high and the rate of stomach emptying rapid, owing to the risk of formation of an anastomotic ulcer. In patients over the age of 50, who will not adhere strictly to medical treatment, a short-circuiting operation may be performed, apart from stenosis. This is because the gastric acidity tends to fall after the age of 50. All septic foci should be eliminated. The after-treatment following gastro-enterostomy should be rigid as regards diet and alkalis, as detailed in the treatment of gastric ulcer (see p. 36).

**Curative.** A strict course of medical treatment, as for gastric ulcer, should be tried. If this fails, operative treatment consists in excision of the jejunal ulcer and undoing the gastro-enterostomy, providing that the original duodenal ulcer has healed.

### Gastro-Colic Fistula

**Etiology.** A gastro-colic fistula may follow the formation of an anastomotic ulcer which has developed after a gastro-enterostomy. In other cases carcinoma of the stomach may spread directly to the colon.

**Clinical Findings.** The principal symptoms are diarrhœa occurring 2 to 3 hours after food and during the night, with eructations having a faecal odour. *On Examination:* There is usually loss of weight. An excess of fat is generally present in the stools, the fat splitting being normal. Further signs of deficient intestinal absorption are a low blood calcium content and a megalocytic anemia. These findings resemble those of sprue, but with gastro-colic fistula there is generally hyperchlorhydria and in sprue hypochlorhydria. The diagnosis is best confirmed by a barium enema.

**Treatment.** This is surgical for cases due to gastric ulcer, but for those due to malignant disease only symptomatic treatment is available.

### Intestinal Diverticula

**Definition.** Pouches in the intestine.

**Etiology.** Diverticula occurring in the small intestine may in some instances have a congenital origin, but usually they are only recognisable

in adult life Meckel's diverticulum is a congenital abnormality of the ileum Diverticula of the large intestine are probably acquired Factors which enter into their causation are —Focal sepsis, especially in the teeth Local inflammatory changes in the intestine, associated with stagnation of its contents A weak spot in the intestinal wall, at the site where the vessels enter and between the bands of longitudinal muscle Overaction of the parasympathetic system Diverticula are slightly more common in males, they occur especially after the age of 40, but are not notably associated with obesity

**Pathology** A congenital diverticulum carries with it all the coats of the intestine The acquired variety herniates through the muscular coat, and consists of the mucous membrane, the serous layer, and an outer fibrous coat may form In the large intestine the diverticula are often extruded into the appendices epiploicæ, so that they are not visible externally until the fat is removed They then appear as bluish black sacs the colour being due to the faecal contents showing through their thin wall Their neck may be constricted so that the contents cannot escape In the intestine diverticula are found occasionally in the duodenum, rarely in the jejunum and ileum and commonly in the large intestine They are most often present in the pelvic and descending colon very rarely in the ascending colon but they may occur in the appendix and rectum They are usually multiple in the large intestine

Three stages are described in their formation 1 A prediverticular stage There is localised weakening of the mucous membrane and hypertrophy of the circular muscle of the intestine 2 Diverticulosis Here developed diverticula are present, but they are not inflamed 3 Diverticulitis Inflammation is present in the neck of the diverticulum and in the neighbouring intestine The lumen of the neck is usually obstructed with faeces

**Clinical Findings** A history of constipation is only obtained in about half the cases

*In the prediverticular stage* there are usually no symptoms unless constipation is present X ray examination with an opaque meal or enema shows an area localised to one part of the intestinal wall where the normal haustra are replaced by a series of convex irregularities These areas may be tender on palpation at the X ray examination *With diverticulosis* there may be no symptoms, or such symptoms as frequency of micturition, pain in the left lower abdomen, intestinal flatulence, diarrhoea or constipation, and discomfort after defaecation may be complained of Abdominal examination yields no information X ray films show the established diverticula, which may remain filled after the intestine has emptied *When diverticulitis is present* the symptoms are more definite, there may be periodical attacks of fever, with leucocytosis and abdominal pain usually in the left lower abdomen A sausage like tumour may be palpable in this site The bowels never feel to be properly emptied, frequency of micturition may occur especially after defaecation The opaque meal or enema reveals the characteristic appearances, the affected portion of intestine is fixed

and thickened and spikes project from the wall like a pallisade. These do not alter in position in serial films although the intestine above and below may contract forcibly. In about 70% of cases of diverticulitis arthritis of the lumbar vertebræ is found by X-rays, and there may be severe pain in the back.

**Differential Diagnosis.** Diverticulitis may be mistaken for left-sided appendicitis, actinomycosis of the intestine, salpingitis or carcinoma. The diagnosis can only be made by a competent radiologist.

**Course and Complications.** The course is usually slowly progressive. Complications include: "Pistol-shot" perforation in which a stercolith is suddenly forced through the diverticulum. Inflammatory perforation, with localised or generalised peritonitis. Intestinal obstruction. Vesical fistula, air and feces appearing in the urine. Vaginal or colic fistulæ. Carcinoma is a very rare sequela.

**Prognosis.** This is good in diverticulosis and the early stages of diverticulitis, providing adequate treatment is given. Death may occur from perforation, obstruction or fistula formation.

**Treatment.** In the acute attacks of diverticulitis the patient should be put to bed, rectal injections of 4 to 6 fl. oz. of warm olive oil are given at night, and a saline colonic washout of 1 to 2 pints of normal saline in the morning. The washout is given as follows: The saline is warmed to 102° F. and run in through a funnel and No. 10 soft rubber catheter. The catheter is injected for 2 to 3 inches into the rectum, with the patient lying in the left lateral position. The funnel is held 12 inches above the anus. The fluid is run in slowly, the patient taking deep breaths. The patient is then assisted into the knee-elbow position and after a few minutes lies in the right lateral position. He should retain the fluid as long as possible. The patient should also take  $\frac{1}{2}$  fl. oz. of paraffin liq. in 2 oz. of milk t.i.d.; the diet should be lacto-vegetarian as detailed below. Operation is required for perforation or obstruction. For lesser degrees of diverticulitis and for diverticulosis or the prediverticular stage, treatment consists in colonic washouts with saline, as above, every other day for several weeks, and later twice a week for several months. These must be adequately given to run round all the colon. The teeth should be X-rayed and all septic ones removed.

*Spriggs' diet* is very efficacious. It is as follows:—

7 a.m.  $\frac{1}{2}$  oz. of liquid paraffin in 2 oz. of warm milk.

8 a.m. Coffee and milk,  $\frac{1}{2}$  oz. of lactose, wholemeal bread and butter with honey or marmalade.

10.30 a.m. Glass of buttermilk, wholemeal bread and butter.

1 p.m. Fish (cooked in any way), butter sauce, salad and dressing, compote of fruit, cream, toast and butter.

4 p.m. Coffee, with milk or cream, marmalade, wholemeal bread or toast and butter.

7.30 p.m. Vegetable soup. Eggs (poached, scrambled or omelette), vegetables, fruit, cream cheese, wholemeal bread and butter. Modifications of this diet can be made according to the taste of the patient. The paraffin should also be given after lunch and dinner, if necessary.

The habitual use of liquid paraffin is deprecated by certain authorities on the grounds that its presence in the rectum prevents complete evacuation, that it interferes with the absorption of vitamins A and D, that it hastens the movements of the contents of the small intestine resulting in incomplete digestion, that it leads to symptoms of indigestion and loss of weight, and that it may also cause pruritus ani.

### Appendicitis

**Definition** Inflammation of the appendix.

**Etiology** The cause is not definitely known. In some cases it may be due to invasion with micro organisms, such as streptococci and the B. coli, rarely with the Streptothrix actinomyces or the Mycobacterium tuberculosis (B. tuberculosis). The organisms enter the appendix from the intestine or from a near by inflamed organ, in some cases they may be blood borne as from a distant focus in the tonsils or teeth. Internal mechanical obstruction from hardened feces (stercoliths or enteroliths), from pips in food or from thread worms or external obstruction from bands may also lead to appendicitis. **Predisposing causes** 1 Age. Chiefly between 10 and 40 years. 2 Sex. Males predominate slightly. 3 Race. Especially common in civilised countries. In some cases a familial incidence can be traced.

**Pathology** On removal the appendix is acutely inflamed, with hyperæmia of the mucous membrane and a dull appearance of the peritoneal coat, or it is enlarged and dilated, or the mucous membrane is ulcerated. The appendix may be filled with pus, or it may be gangrenous, or perforated, with local or general peritonitis. In chronic cases it may be distorted and bound down by adhesions and the lymphatic glands at the appendix root and in the ileo cæcal angle enlarged. In actinomycosis the appendix and cæcum are frequently involved and the disease spreads to the peritoneum, abdominal wall and liver.

**Clinical Findings** Acute appendicitis is a surgical complaint, and will not be considered in this book. Chronic appendicitis frequently causes difficulty in diagnosis. It may follow an acute attack of appendicitis or more often develop insidiously. The patient complains of periodical attacks of pain in the right lower abdomen or around the umbilicus, of a griping nature (appendicular colic) and there may be nausea, vomiting, constipation or diarrhoea. In the intervals the patient is usually well. In other instances he suffers from chronic ill health (appendicular dyspepsia), with such symptoms as recurring nausea, anorexia, constipation, fulness after meals and pains in various parts of the abdomen. A momentary sharp stabbing pain may be felt in the right iliac region on walking. Frequency of micturition may be noticed if the appendix irritates the bladder. A focus of infection in the appendix may be the causative factor in such lesions as gastric or duodenal ulcer, cholecystitis or infective arthritis.

**On Examination** In a well marked case tenderness can be elicited on firm pressure over McBurney's point (the junction of the outer and middle thirds of a line drawn from the umbilicus to the right anterior



superior iliac spine). The pain elicited may be referred to the umbilicus. In other cases this tenderness can only be demonstrated when the appendix is filled with barium and palpated while it is visualised with X-rays. There is often slight rigidity of the right lower rectus muscle. Occasionally the appendix lies in an abnormal situation, such as in the pelvis, when it may be tender on rectal or vaginal examination, or it may be retro-cæcal, or under the right costal margin, or even on the left side of the abdomen, if the viscera are transposed. The temperature is at times slightly raised. In a doubtful case inflation of the large intestine through a rectal tube may cause pain in the appendix region (Bastedo's sign). Ulceration of the mucous membrane of the appendix will account for the presence of occult blood in the fæces.

**Differential Diagnosis.** Chronic appendicitis must be differentiated from intestinal colic, intestinal angio-neurotic oedema, pyelitis, a gastric or duodenal ulcer, chronic cholecystitis, biliary colic, renal colic, a growth near the cæcum, ileo-cæcal tuberculosis, regional ileitis, chronic salpingitis or oophoritis, or enlarged glands (usually tuberculous) in the right iliac fossa. The urine should always be examined microscopically to exclude the presence of organisms. An X-ray examination of the abdomen will help to exclude a stone in the gall-bladder or kidney or calcified abdominal glands. An opaque meal helps to eliminate the possibility of a gastric or duodenal ulcer, and a tender appendix may be palpated under the screen as mentioned above. If the appendix fills with barium, it is usually possible to determine whether or not it is fixed by adhesions; if the appendix does not fill, there is evidence that the lumen is occluded, and further ileal or cæcal stasis is suggestive of chronic appendicitis. A cholecystogram helps to exclude cholecystitis. It is often impossible to diagnose ileo-cæcal tuberculosis without an operation, but in some cases pulmonary tuberculosis is also present. A lump may be palpable on abdominal examination, which can be mistaken for carcinoma.

**Course and Complications.** Chronic appendicitis is characterised by recurrent attacks of pain or dyspepsia; ultimately an attack of acute appendicitis may develop. In other cases the attacks gradually cease, perhaps owing to permanent obliteration of the lumen. Complications include: Abdominal adhesions, perforation, peritonitis, gastric or duodenal ulceration, cholecystitis and colitis.

**Prognosis.** This is always uncertain.

**Treatment.** A chronically inflamed appendix which is causing symptoms should be removed surgically, unless an operation is contra-indicated by some other coexistent disease, such as pulmonary tuberculosis. Appendicular colic may sometimes be relieved by the administration of tne, belladon. m. 10 to 15 t.d.s. a.c. The treatment of actinomycosis is described on p. 586.

### Colitis

**Definition.** The term colitis includes inflammation and degeneration of the mucous membrane of the colon, and conditions associated with excessive secretion of mucus. The following varieties are described:

Acute catarrhal    Chronic catarrhal    Mucous or muco membranous  
Ulcerative

### Acute Catarrhal Colitis

**Etiology** Acute catarrhal colitis may occur as a part of acute gastro enteritis (see p 22) sometimes due to an infection with the *Bacterium friedlander* (*pneumobacillus*) to influenza or to drugs such as colchicum or it may be associated with such diseases as enteric fever, intestinal tuberculosis dysentery an intestinal neoplasm or uremia

**Clinical Findings** The patient complains of diarrhoea and the stools contain mucus and at times bright blood There is usually some abdominal colic and the temperature may be raised

**Treatment** This varies with the cause and is as described under the various diseases with which it is associated

### Chronic Catarrhal Colitis

**Etiology** Chronic catarrhal colitis may be a sequela of acute catarrhal colitis or of dysentery or it may result from the misguided habitual use of purgatives

**Clinical Findings** The motions are loose, frequent and contain some bright blood and mucus

**Treatment** This again varies with the cause In general a diet should be taken which is free from irritants (see p 52) Liquid paraffin m 60 should be taken t d s p c and if there is pain due to spasm of the colon a mixture containing the belladonna m 7 to 10 t d s a c usually affords relief An abdominal belt should be worn as the condition is made worse by chill

### Muco-membranous Colitis

(*Muco membranous Colic    Mucous Colitis*)

**Etiology** Mucous colitis is believed in some cases to be due to over activity of the bulbo-sacral autonomic nerves which supply motor fibres to the large intestine Such a condition is analogous to asthma, there being an increased output of mucus from the glands and muscular spasm In other cases of mucous colitis there are associated disorders, of these the most important are abdominal adhesions diverticulitis visceroptosis an intestinal growth and chronic appendicitis *Pre disposing causes* 1 Age Usually over 20 2 Sex Females predominate 3 Nervous instability is often present

**Pathology** Often no changes are found post mortem in the mucous membrane of the colon but a true inflammatory or granular colitis may be present

**Clinical Findings** The patient is usually a middle-aged woman who complains of passing mucus in the motions She is frequently constipated but there may be periodical attacks of diarrhoea In some cases there are attacks of constipation followed by abdominal colic after which mucus is passed There may also be abdominal discomfort or pain generally in the left iliac region

*On Examination:* The patient is usually, but not invariably, of a nervous type, introspective, with her thoughts concentrated on her motions. She is thin and the tongue is somewhat furred. The descending colon is palpable if it is in spasm or if the patient is constipated, and it may be tender. The motions are usually constipated and the mucus may be passed as long whitish strips, or as a tubular intestinal cast, or in balls. If the rectum is involved there is frequent tenesmus, with passage of mucus. Some blood is at times present in the motions. The motions may also contain yellowish brown granules of intestinal sand, composed of calcium salts of palmitic, stearic and phosphoric acids, with urobilin. There may be a low grade of pyrexia, such as a temperature of 99° to 100° F. in the evenings. Sigmoidoscopy: An excess of mucus is seen covering the mucous membrane; no ulceration is present, but in some cases the mucous membrane appears inflamed or polypoid.

*Differential Diagnosis.* Mucous colitis has to be differentiated from carcinoma of the colon, diarrhoea due to achlorhydria, and ulcerative or catarrhal colitis. The diagnosis is established by the history of the mucus in the stools, the sigmoidoscopic and barium enema findings, the latter showing no evidence of a growth.

*Course and Complications.* The disease is very chronic, and often is present for many years. Neuritis, arthritis and pyelitis may occur as complications.

*Prognosis.* Cure is difficult to effect, and a relapse is not infrequent after cases have much benefited by a course of rigid treatment.

*Treatment.* The patient should be put to bed in severe cases, or for the purpose of investigation. The constipation must be relieved first by rectal injections of 2 to 4 fl. oz. of warm olive oil, which are later increased up to 10 fl. oz. This is retained during the night and followed by an enema of warm normal saline in the morning. Liq. paraffin m. 60 to 240 is also given t.i.d. p.c. Intestinal spasm is combated by a mixture containing Tnc. belladon. m. 5 to 10, sod. brom. gr. 5, sod. bicarb. gr. 10, sp. chlorof. m. 7, aquam ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  t.d.s. a.c.

The diet must not be irritating; no foods leaving residue should be eaten; no salads, skins of fruit or fish, or pips must be taken.

### Ulcerative Colitis

*Definition.* A chronic affection of the colon, characterised by ulceration of its mucous membrane. Ulceration due to dysentery, tuberculosis, syphilis and enteric fever is not included under this heading.

*Etiology.* The cause is not known; by some it is believed to be due to dysentery or pseudo-dysentery bacilli, by others to an avitaminosis, and by others to unknown chemical substances present in food. *Predisposing causes:* Outbreaks have occurred in institutions such as asylums. Adults are usually affected.

*Pathology.* The ulcers are superficial and chiefly situated in the pelvic colon.

*Clinical Findings.* The patient is usually an adult, who complains

of diarrhoea which gradually becomes worse. There is often abdominal discomfort or pain, in the left iliac region. If the lesions are low down there is also tenesmus.

**On Examination** The patient is usually wasted, and there may be a low grade pyrexia of  $99^{\circ}$  to  $100^{\circ}$  F. The stools are loose and contain mucus, bright blood and at times some pus. They may occur up to 10 or 20 times in the 24 hours. Sigmoidoscopy reveals a swollen and red mucous membrane, with a mucous exudate. Superficial ulcers may be seen, especially on swabbing away the mucus. In advanced stages X-ray examination with a barium enema shows the colon as a rigid, narrow tube, the haustrations usually being absent.

**Differential Diagnosis** Ulcerative colitis must be differentiated from a growth in the colon, from mucous colitis, dysentery and pernicious anaemia. If there is a growth it may be seen with the sigmoidoscope, or if situated higher up, mucus and blood may be seen coming down the colon, the lower part of the mucous membrane appearing healthy. In chronic dysentery the bacilli can usually be isolated from a swab taken from an ulcer, through the sigmoidoscope. The blood count serves to differentiate pernicious anaemia.

**Course and Complications** The disease tends to run a very chronic course. Complications include polyposis or stricture of the colon, peritonitis and arthritis.

**Prognosis** This is usually grave, prolonged treatment is necessary to effect a cure, and even then relapse is liable to occur.

**Treatment** The patient should be put to bed. The diet is as for mucous colitis. Sulphaguanidine should be given a trial, as for dysentery (see p. 686). A starch and opium enema (starch gr 60, water fl oz 2, and the opium 30) should be given daily, until the motions are reduced to about four a day. Rectal injections of cod liver oil should then be given, using first fl oz 2 every morning and increasing to fl oz 8. When the patient has become accustomed to retain them for several hours the injections should be given in the evening. If this fails, colonic washouts (see p. 48) should be given every other day with Argylol (argent proteinas mite B.P.C.)  $\frac{1}{2}\%$ , or with tannic acid gr 1 to 1 fl oz of water, using 20 to 30 fl oz, or with hypertonic saline (sodium chloride gr 120 to water 1 pint). For the anaemia iron should be given in large doses, such as ferri et ammon cit gr 30 to 60 t.d.s. p.c. (see p. 492), and blood transfusions may be required. If this fails, surgical treatment should be considered. This should not be delayed until the patient is very debilitated. Several varieties of operation have been recommended. In some cases appendicostomy, followed by daily colonic lavage from above, is satisfactory, more often a terminal ileostomy is the operation of choice, and this, in the majority of cases, has to be permanent.

### Intussusception

**Definition** Telescoping of the intestine. There are two clinical varieties. Acute and chronic.

### Acute Intussusception

**Etiology.** Acute intussusception results from irritation of the intestine and muscular imbalance in its walls. The irritation may be due to hard feces, worms or a polypus. *Predisposing causes:* 1. Age: Usually infants. 2. Sex: Males predominate.

**Pathology.** The upper portion of the intestine is invaginated into that below. There are three layers, the inner, entering or intussusceptum, the returning or middle, and the outer, ensheathing or intussusciens. The mesentery enters with the intestine, and compression of the vessels may lead to inflammation, gangrene, or rupture of the intestine. There are four anatomical varieties. 1. The ileo-cæcal. This is the most common, the ileo-cæcal valve and the ileum enter the colon. 2. The enteric. Here one portion of small intestine enters another. 3. The colic. The colon is invaginated. 4. The ileo-colic. The ileum passes through the ileo-cæcal valve, and then the ileum, ileo-cæcal valve and cæcum pass into the colon.

**Clinical Findings.** The infant is suddenly taken ill with abdominal colic. He may then vomit. The characteristic feature is the loose actions of the bowels; there is tenesmus and passage of odourless mucus and blood.

*On Examination:* In the ileo-cæcal variety, a "sausage-shaped" tumour may be felt in the upper part of the abdomen or in the left iliac region, whereas the right iliac region feels empty (*signe de Dance*). The temperature falls, but the pulse is frequent.

**Differential Diagnosis.** Acute intussusception is most likely to be confused with Henoch's purpura (see p. 521). Careful search must be made for purpuric spots in the skin. If found, no operation must be performed. In acute colitis the motions have a fæcal odour, and no tumour is palpable.

**Course and Complications.** Collapse and death ensue unless the intussusception is reduced.

**Prognosis.** This depends upon efficient treatment.

**Treatment.** This is surgical.

### Chronic Intussusception

**Etiology.** Chronic intussusception occurs in adults, and is usually associated with a simple or malignant growth of the large intestine.

**Clinical Findings.** The patient suffers from attacks of abdominal colic, with diarrhœa, the passage of blood and mucus, and at times vomiting.

*On Examination:* A tumour is palpable in the abdomen in some cases, or the invaginated intestine may even protrude from the anus.

**Differential Diagnosis.** A barium enema may disclose the nature of the trouble.

**Course and Complications.** The intussusception may unravel itself spontaneously, but usually it tends to recur and, after persisting for several months, results in obstruction or perforation.

**Prognosis.** The outlook is unfavourable.

**Treatment.** This is operative.

### Intestinal Obstruction

**Definition** Obstruction to the passage of faeces through the intestine

**Etiology** The obstruction may be due to 1 *Causes in the lumen or wall of the bowel* These include —A volvulus An intussusception A growth Impacted faeces A stricture due to syphilis or following dysentery A foreign body, such as a gall stone which has ulcerated through into the duodenum (the obstruction then usually occurs in the terminal part of the ileum) Other foreign substances such as masses of thread worms or a hair-ball Paralytic ileus, in which there is no mechanical obstruction, thus may follow an abdominal operation or complicate acute appendicitis, peritonitis or torsion of the pedicle of the spleen Regional ileitis (Crohn's disease) in which there is cicatricial stenosis usually of the last 12 to 14 inches of the ileum, following a chronic inflammatory condition of unknown origin Multiple fistulae may be present, communicating either with the large intestine or tracking through the anterior abdominal wall Any part of the ileum may be involved, and in some cases the adjacent lymph glands have been shown to contain giant cells although tubercle bacilli have rarely been demonstrated

2 *Causes outside the bowel* Strangulation or obstruction may result from —1 A band This may be due to plastic peritonitis or follow a laparotomy The band may form between two portions of the mesentery, or between the mesentery and an abdominal viscus or inflamed gland 2 A cord This may pass from the omentum to an abdominal viscus or to the abdominal wall 3 Meckel's diverticulum may be attached to the umbilicus or to some viscus in the abdomen or to the mesentery 4 The bowel may be strangulated or obstructed in an internal hernial orifice, in the foramen of Winslow, or in a diaphragmatic hernia

In strangulation the flow of blood through the vessels of the bowel is interfered with, but the passage of faeces is not prevented

**Pathology.** The portion of bowel above the obstruction is dilated, the wall is hyperæmic and the contents are fluid consisting of an exudate from the wall and vessels and many bacteria Histamine may form in the contents, and on absorption give rise to the symptoms of shock At the site of the block the intestine is also distended, its coat becomes purplish in colour, and it may be gangrenous and sloughing, with the vessels thrombosed It usually contains blood stained fluid and gas Below the obstruction the bowel is empty, contracted and pale Peritonitis may occur around the site of the obstruction The obstruction may occur acutely, as with a band, volvulus intussusception or incarceration in a hernial orifice, or more gradually, as with a growth in the large intestine, where hardened faeces may prove the last factor producing a total block The "faecal" vomiting is probably not due to antiperistalsis and regurgitation of faeces, but to an increased exudate into the bowel, the fluid contents well up to the stomach, and are vomited without effort

**Clinical Findings** With acute obstruction the patient is suddenly

taken ill with severe tearing and colicky pains in the abdomen, which double him up; he becomes prostrated and collapsed. Vomiting sets in and there is great thirst. The vomit at first consists of the gastric contents, later of bile, and finally it is of the faecal "regurgitant" type. A motion may be passed early in the attack, but after this no faeces or flatus escape from the rectum.

*On Examination:* The patient is seen to be very ill; he is pale, the skin is moist, the tongue furred and later dry, the temperature is sub-normal, and the pulse frequent. The abdomen is generally distended, but it is not always tender. A tumour may be felt, as with a volvulus, or visible peristalsis may occur from time to time. The higher up in the intestine that the obstruction occurs the more acute are the symptoms. The blood urea and non-protein nitrogen figures are raised. In chronic obstruction the onset is more gradual, and diarrhoea may at first alternate with constipation, then absolute constipation with symptoms of obstruction sets in. The patient should be given a turpentine enema (turpentine fl. oz.  $\frac{1}{2}$  and soap and water fl. oz. 10.) This is returned clear, without any force and without the passage of flatus in the presence of obstruction. A direct X-ray of the abdomen will show excess of gas in the large intestine if this is obstructed. The presence of gas in the small intestine is also a sign of obstruction there, and in some cases fluid levels are seen.

*Differential Diagnosis.* Intestinal obstruction must be diagnosed from other acute abdominal lesions, such as a perforated gastric or duodenal ulcer, acute appendicitis, mesenteric thrombosis, and acute pancreatitis, and from such conditions as biliary or renal colic, lead colic and abdominal crises in tabes or encephalitis lethargica. All the hernial orifices should be examined. The history of the case, the results of the clinical examination and the inability of the patient to pass faeces or flatus usually enable the diagnosis to be established, before the stage of faecal vomiting is reached.

*Course and Complications.* Unless relieved surgically, intestinal obstruction is usually permanent. Complications such as perforation and peritonitis may occur.

*Prognosis.* Death occurs in a few days, unless the obstruction is relieved.

*Treatment.* An operation should be performed without delay to relieve the obstruction if it is not paralytic in origin. Morphine should not be given until the diagnosis has been made. For paralytic ileus stimulant treatment is first applied. One mil. of Pituitrin (ext. pit. liq. B.P.) is injected intramuscularly followed in about a quarter of an hour by a glycerin enema (glycerin and water, of each fl. oz.  $\frac{1}{2}$ ). If no flatus is passed further stimulant treatment should be applied, such as the intramuscular injection of a mixture of acetyl-choline (B.P.C.) 0.2 G., Pituitrin 0.5 mil. and physostigmine salicylate gr.  $\frac{1}{60}$ , every hour for four doses. This is followed by an enema of ox bile fl. oz. 2 and normal saline fl. oz. 4, a simple enema being given half an hour later. If this fails, it is best to abandon stimulant treatment and inject subcutaneously morphin. sulph. gr.  $\frac{1}{6}$ , apply heat to the abdomen.

by means of an electric cradle and give an intravenous drip injection of 1 pint of normal saline containing 5% dextrose. To relieve vomiting a Ryle's stomach tube should be passed and suction applied, either by a syringe or by attaching the tube to an inverted bottle containing water, suspended above the patient, whereby syphonage can be established.

### Intestinal New Growths

**Etiology** The cause of new growths is not known. Carcinoma is usually a disease of adult life, being very uncommon in infants and children. The sexes are equally affected. *Varieties* An intestinal tumour may be simple, such as an adenoma, polypus, myoma, lipoma or hæmangioma, or more commonly a malignant tumour such as a carcinoma. Sarcoma is rare.

**Pathology** Polypi may be multiple, occurring in the large intestine. Hæmangiomata also may be multiple. Carcinoma is found in the large intestine, in the following sites, in order of frequency: The rectum, pelvic colon, cæcum, transverse colon, splenic flexure, ascending colon, hepatic flexure and descending colon. It is rarely met with in the appendix and small intestine. Secondary deposits occur comparatively late in the disease.

**Clinical Findings** Hæmangioma of the small intestine may give rise to recurrent attacks of mæna with severe anæmia but usually no pain. The patient suffering from a malignant growth is usually an adult over the age of 40. The first symptom may be the onset of persistent constipation or of periodical attacks of diarrhœa or of alternating constipation and diarrhœa where the bowels previously have acted very regularly. The patient may also notice abdominal discomfort or fulness, a feeling of general illness, and loss of weight. The appetite may remain good. He may complain of bleeding from the rectum or of a frequent desire to go to stool, if the growth is low down in the rectum.

**On Examination** In the early stages often no localising signs can be found, as the growth enlarges it may become palpable on abdominal or rectal examination. Some pallor and cachexia may also be apparent. There may be slight and irregular fever. The motions may contain macroscopic or occult blood, and mucus or pus. In some cases ribbon-shaped motions are passed due either to compression by the growth or to reflex anal spasm. With the sigmoidoscope the lower 10 inches of the alimentary tract can be inspected, and this examination should never be omitted in a doubtful case. A barium enema is more likely to reveal a growth in the large intestine, than is a barium meal. In more advanced cases evidence of secondary deposits may be found in other organs, such as the liver, and there may be ascites.

**Differential Diagnosis** Hæmangioma is not often diagnosed even at laparotomy. It should always be borne in mind in cases of recurrent mæna and anæmia. Telangiectases may be seen in the serous coat of the affected part of the intestine. The diagnosis of an intestinal malignant growth depends upon the combined results of a manual examination, sigmoidoscopy, the barium enema and the occult blood



test. Other conditions which require exclusion are: Hæmorrhoids, simple constipation, redundant loops of the colon, colitis, diverticulitis, actinomycosis, Crohn's disease, a chronic appendix abscess, a gastric neoplasm, enlargement of the spleen, enlargement of the gall-bladder or kidney, hyperplastic tuberculosis of the cæcum, tuberculous peritonitis, and ascites due to other causes.

**Course and Complications.** The course is slowly progressive; secondary deposits occur in the abdominal glands, peritoneum, and other organs, such as the liver and brain. Intestinal obstruction or perforation may occur as complications.

**Prognosis.** This is fatal, unless the growth can be removed before metastases have formed. Death usually occurs in 1 to 2 years from the appearance of symptoms.

**Treatment.** In cases of hæmangioma the affected portion of the intestine should be resected. Simple tumours may be removed surgically and malignant ones either treated by operation or by radio-therapy. A colostomy may be required as a palliative measure to relieve obstruction.

### *Hirschsprung's Disease*

*(Achalasia of the Pelvi-rectal and Anal Sphincters. Idiopathic Dilatation of the Colon. Megacolon)*

**Definition.** Dilatation and hypertrophy of the colon arising without apparent cause.

**Etiology.** The enlargement of the colon may result from a disorder of the neuro-muscular mechanism controlling the pelvi-rectal or anal sphincters, comparable with achalasia of the cardia (see p. 18). It is considered to be a congenital anomaly. The cases which manifest themselves in adult life (megacolon) have probably a similar origin, but are aggravated by constipation. Some cases occur in association with steatorrhœa (see p. 60). The disease is more common in males than in females.

**Pathology.** The descending and pelvic colon are chiefly affected, but the rectum is often involved in adults. The colon is much enlarged and may measure 12 inches in diameter; the muscular coats are hypertrophied and stercoral ulceration of the mucous membrane may be seen. In a severe case the contents of the colon may weigh over 3 stones.

**Clinical Findings.** The condition may be noticed soon after birth, the infant being extremely constipated and the abdomen becoming distended. In other cases the distention is not apparent until adult life. The patient further complains of ill-health, occasional vomiting, abdominal colic, and some dyspnœa from upward displacement of the diaphragm. The bowels may not be opened for several months, but attacks of diarrhœa sometimes occur.

**On Examination:** The abdomen may present a ballooned appearance, being either diffusely distended or more so on the left side. Visible peristalsis may be present. If the rectum is involved, digital examination shows that it is full of hard fæces. A barium enema may reveal an enormously distended colon.



FIG. 1. A CHILD SUFFERING FROM CELIAC DISEASE, AGE 26 MONTHS, WEIGHT 18 LBS. 4 OZS., HEIGHT 30½ INCHES.

steatorrhœa and sprue. In Hirschsprung's disease constipation has usually been noted shortly after birth, fatty stools may occur with lacteal obstruction; in sprue the appetite is usually good, and the tongue is sore, and in pancreatic steatorrhœa there is no interference with growth, and the faecal fat is chiefly unsplit.

**Course and Complications.** The course is usually prolonged. Complications such as rickets, a generalised but slight œdema, tetany or purpura may occur.

**Prognosis** There is a tendency to recovery in the course of 2 or 3 years, but death may result from an intercurrent disease. The expectation of normal development as regards height and sexual maturity is always uncertain.

**Treatment.** Fat and carbohydrate must be restricted in the diet. Dried milk, white of egg, orange juice, green vegetables, chicken broth, meat and bananas up to 6 or more a day should be given. Vitamins A and D should be supplied as in the form of Radiostolcum capsules (liq vitamin A et D conc B P Add) m 3, 1 t d s, to prevent the development of rickets. In addition bile salts, such as sodium glycocholate gr 1 and sodium taurocholate gr 1, should be given t i d in a capsule and liq magnes bicarb m 30 t i d to assist in the saponification of the fats.

### Cœliac Disease in Adults

*(Idiopathic Steatorrhœa Gee's Disease Non tropical Sprue.  
Gee Thaysen Disease)*

**Definition** A disease of adults characterised by steatorrhœa and disturbances of calcium and phosphorus metabolism.

**Etiology** The cause is unknown. It is believed that there is an abnormal absorption of foodstuffs from the small intestine, chiefly of lipoids, and to a lesser degree of calcium, phosphorus, hæmopoietic substances and vitamins. *Predisposing causes* 1 Age Adolescents and adults 2 Sex. Females predominate slightly.

**Pathology.** Post-mortem examination shows no characteristic change.

**Clinical Findings** A history can often be obtained of diarrhœa or of rickets in infancy. The patient complains of such symptoms as diarrhœa, weakness, skin eruptions, muscular cramps, pains in the bones, or bony deformities, especially knock knee.

**On Examination** The growth is stunted and the abdomen is often protuberant. The patient presents an infantile appearance. Skin lesions may be seen, such as areas of erythema, pigmentation or exfoliative dermatitis, especially on covered portions of the skin. Spontaneous fractures may be found in various bones, genu valgum or varum is common and the bones show osteoporosis. The fingers may show parrot beak clubbing. In some cases the colon is distended (megacolon). Changes in the small intestine have also been found by X ray examination, such as obliteration of the markings of the valvulæ conniventes, distention of the gut and segmentation of the contained barium into clumps. The blood. There is often an *anæmia*, either hypochromic,

megalocytic or erythroblastic in type. The serum calcium is often low, the plasma phosphorus is usually low but may be normal or high, and the phosphatase content rises with the activity of the bony changes. The blood sugar is often low, and the sugar tolerance curve flat. The stools are either fatty or appear normal to the naked eye, but chemical examination reveals an excess of fat (40 to 70%). The fats are well split. Increased muscular irritability may be demonstrated by Chvostek's or Trousseau's sign (see p. 661). Opacities in the lens are revealed by the slit lamp in some cases.

**Differential Diagnosis.** Cutaneous diseases such as psoriasis or exfoliative dermatitis may be diagnosed, without the true nature of the disease being recognised. In other instances tetany is the predominating feature. The clinical findings resemble those of sprue (see p. 692).

**Course and Complications.** The course is usually progressive unless adequately treated. Intercurrent infections, such as bronchopneumonia, may ensue.

**Prognosis.** The welfare of the patient can be very materially improved by adequate treatment.

**Treatment.** The patient should be given a fat-poor diet. Carbohydrates should also be restricted if there is much flatulence. Calcium should be administered in the form of calcium lactate gr. 40 t.i.d. and vitamins A, B<sub>1</sub>, B<sub>2</sub>, C and D should also be given. Suitable preparations are: Vitamins A and D, Adexolin capsule (liq. vitamin A et D conc. B.P. Add.) m. 3, 1 t.d.s., B<sub>1</sub>, Benerva tab. (aneurin. hydrochlor. B.P. Add.) mg. 3, 1 daily, B<sub>2</sub>, riboflavin tab. mg. 1, 1 daily, and C, Redoxon tab. (ascorbic acid tab. B.P. Add.) mg. 50, 1 daily. Hypochromic anæmia and erythroblastic anæmia should be treated with iron, such as ferri et ammon. cit. gr. 15 to 30 t.d.s. or Bland's pill (pil. ferri carboratis B.P.), gr. 30 t.d.s., and megalocytic anæmia with Marmite,  $\frac{1}{2}$  oz., daily or the intramuscular injection of liver. Two mls of a preparation such as Hepatex I.M. should be given every other day. This is also thought to aid fat absorption.

### Constipation

**Definition.** Delay in the passage of *fæces* through the intestines or delay in their evacuation.

**Etiology.** The following varieties are described: *Intestinal constipation*: There is delay in the passage of the *fæces* through the intestines, usually in the colon. *Dyschezia*: Here the intestinal transit time is normal, but the *fæces* accumulate in the pelvic colon and rectum. "*Greedy colon*": Excessive absorption reduces the bulk of the *fæces*.

**Intestinal Constipation.** The delay may be due to: 1. The small bulk of *fæces*, as would result from insufficient food, from a diet poor in residue (such as cellulose), from insufficient fluid intake, from excessive fluid loss as in diabetes mellitus or insipidus, from excessive sweating, or from excessive absorption as in "*greedy colon*."

2. A large bulk of *fæces*, as in heavy eaters, causing difficulty in propulsion.

tumour, masses of hard faeces amyloid degeneration of the intestines, and leukæmic infiltration of the intestines Cirrhosis of the liver with portal congestion, passive hyperæmia secondary to heart failure, and colitis of various types (see p 50)

4 *Mesenteric Causes* *Tabes mesenterica*, and obstruction of mesenteric lacteals causing chylous diarrhœa

5 *Deficiency of Pancreatic or of Biliary Secretion*

6 *Carbohydrate Fermentation* Associated with excessive intake of carbohydrate foods and deficient exercise

7 *Hypothyroidism*

**Clinical Findings** These must vary with the underlying cause In henteric diarrhœa the patient is usually of a nervous type, a child or an adult, a formed motion may be passed after breakfast, but during the morning there are several loose motions There is often great urgency, so that immediate defæcation cannot be restrained In the afternoon a similar state of affairs recurs There is usually no diarrhœa during the night Gastro colic fistula is described on p 46 Hypo thyroidism can be demonstrated by a BMR determination The clinical findings with the other causes of diarrhœa are considered under their respective headings

In all cases of diarrhœa the stools should be examined by naked eye as regards colour, appearance, presence of mucus, blood, froth, etc., and in the laboratory for the presence of undigested food residue, the fat and bile content, and the presence of pathological organisms

**Treatment.** In henteric diarrhœa all food leaving an irritating residue should be avoided, food should not be taken very hot or ice cold A mixture is given containing *Enc belladon m 5 to 10, sod brom gr 5, sp chlorof m 5 aquam ad fl oz ½ Fl oz ½ tds a c* The dose of belladonna is varied according to the result produced In achlorhydria the use of acid hydrochlor dil m 30 to 90 tds p c in half a glass of water, often checks the diarrhœa In hypothyroidism, *thyroideum gr ¼ to 1* daily will often cure the diarrhœa The treatment of the other forms of diarrhœa is considered under the respective headings of the causative diseases

### Visceroptosis

(*Glénard's Disease Splanchnoptosis Enteroptosis Chronic Intestinal Invalidism*)

**Definition** A syndrome characterised by downward displacement of the abdominal viscera with disturbed function of the affected organs and of the mind

**Etiology.** Various theories have been propounded to account for the visceral displacement These include A congenital malposition, the pressure of corsets chronic peritonitis at the mesenteric root with contraction of newly formed connective tissue, a pendulous abdomen with excess of intra abdominal fat, a long narrow thorax with wasting and loss of intra abdominal fat, weakness of the abdominal and pelvic muscles with lowering of the intra abdominal pressure (this is, however, normally a negative pressure), intestinal stasis causing dropping of the

colon and secondary deficient hepatic function (hepatism), faulty postural habits, thoracic breathing and weakness of the transversalis abdominalis muscles. X-ray examinations with an opaque meal have shown that the stomach may lengthen, but it does not "drop," the upper point of attachment remaining fixed. The stomach empties by muscular contractions and not by gravity. The lowest point of the greater curvature of the stomach lies below the umbilicus (inter-iliac line) in the erect position in about 80% of healthy people, the lesser curvature is below this plane in about 20% of healthy people, and the pylorus is below the transpyloric plane in about 70% of such people. The transverse colon may be low without any accompanying symptoms. The symptoms of visceroptosis cannot be entirely attributed to an associated constipation, for intestinal autointoxication appears rather to be associated with diarrhoea than with constipation. Some of the symptoms may be due to vascular disturbances with low blood pressure and splanchnic dilatation. It is evident that displacement of viscera alone does not produce the symptoms of visceroptosis. There must also be disturbance of function. The chief physical defects producing the syndrome are a poor muscle tone, postural errors, and a disturbance of nutrition, either obesity or wasting. There may be an associated neurasthenia. *Predisposing causes:* 1. Age: Usually between 20 and 40. 2. Sex: Females predominate, about 10 to 1. 3. Abdominal distention, due to repeated pregnancies, but the abdominal wall may be very lax without any associated symptoms. 4. Severe illnesses, causing wasting.

**Pathology.** The kidneys, spleen, liver and cæcum may be unduly mobile. The colon may lie low in the abdomen, and redundant loops be present.

**Clinical Findings.** The patient is usually a young or middle-aged woman. A history can generally be obtained of progressive ill-health. The ability to carry out physical or mental work deteriorates, interest and concentration flag easily. Various abdominal symptoms are present, such as constipation, intestinal or gastric flatulence, discomfort and fulness after meals, a dragging sensation relieved by lying down, pain in the epigastrium, around the umbilicus, or in either iliac or hypochondriac regions. There may also be pain or aching in the lumbar regions or over the sacro-iliac joints, and neurasthenic symptoms. The patient is often susceptible to cold and the circulation is poor in the hands and feet. Rheumatic pains may be felt in various parts of the body.

**On Examination:** The patient is generally thin, with a tendency to kyphosis in the dorsal region, the chest is long and narrow, and the subcostal angle acute. On standing the abdominal wall may be seen to bulge below the navel, and the tone of the abdominal muscles is poor. When the patient lies the kidneys may be found to be mobile, the pulsation of the aorta is easily felt, a stomach splash is, in some cases, elicited more than 4 hours after the last meal, and the contents of the pelvic colon may be palpable. The blood pressure is usually low, such as 100/68 mm. Hg. In other cases the patient may be squarely built, with

a pendulous abdomen. A barium meal will reveal the situation of the stomach and intestines, the rate of passage of the meal through the alimentary tract, and the presence and type of constipation.

**Differential Diagnosis** A diagnosis cannot be made until every effort has been taken to exclude other causes for the patient's symptoms. These include chronic appendicitis or cholecystitis, a gastric or duodenal ulcer, abdominal adhesions after operations, chronic peritonitis, and a gastric or intestinal growth. Special examinations include a test meal, barium meal and enema, cholecystogram and investigation of the faeces for occult blood.

**Course and Complications.** The course is insidious and progressive, and, unless adequate treatment is given early, the patient gradually develops into a chronic intestinal invalid. Complications include. Duodenal ileus (see p 44), mucous colitis (see p 51), Dietl's crises (see p 178), and torsion of the pedicle of the spleen.

**Prognosis** This is unfavourable as regards complete restoration to health, although the condition is not fatal.

**Treatment Prophylactic** Children should be trained to adopt a correct bodily poise, exercises should be given to strengthen the abdominal muscles and expand the chest. Any tendency to constipation should be corrected. Women should remain at least 10 to 12 days in bed after childbirth.

**Curative Rest** The treatment should be started with a complete rest in bed for 2 weeks, with the foot of the bed elevated 6 to 9 inches on blocks. The patient should then gradually get up and about again, but only a restricted amount of exercise should be taken. Exercises should be initiated during the rest treatment and continued subsequently. The exercises detailed by MacMahon (*Lancet*, 1925, 1, 108) may be used. The costal angle is expanded by inspiratory breathing to elevate the lower ribs, and the abdominal muscles strengthened by active contractions, with the patient supine. An abdominal support, such as the Curtis belt, may give a feeling of relief when the patient is up, but should be discontinued as soon as the abdominal tone increases. The patient should always lie down and push the abdominal viscera up with the hand when putting on the support.

**Diet** An ordinary mixed diet is the best. If there is much flatulence or gastric distention, the meals should be taken dry and excess of carbohydrate avoided, as for flatulent dyspepsia (see p 27), but at least 3 pints of fluid should be taken in the 24 hours, apart from meals. The patient should lie down on the right side for half an hour after meals.

**Drugs** Sedatives such as bromides should be given to calm the nervous system and aid sleep. They may be combined with Taka-Diastase if there is flatulence. Thus Taka Diastase Liq m 60, sod brom gr 5, sod bicarb gr 10, tnc card co m 30, sp chlorof m 7, infus gent co rre ad fl oz  $\frac{1}{2}$ . Fl oz  $\frac{1}{2}$  ex aqua t d s p c. For constipation, a mixture of Cascara Evacuant m 30 to 90 to which is added water fl oz 1 and paraffin liq fl oz  $\frac{1}{2}$  should be taken at night. An attempt should also be made to improve the mental outlook of the patient and her reaction to external stimuli.

## THE PANCREAS

**Introductory.** Affections of the pancreas may give rise to very diverse clinical and laboratory findings, according to their nature and to the disturbance of pancreatic function. The effects produced may be grouped as follows:—

**Disturbances of Internal Secretion.** These may be demonstrated by: 1. Glycosuria, deficiency of insulin results in diabetes mellitus. 2. Loewi's adrenaline eye test consists in placing 2 drops of a 1/1000 adrenaline solution into the conjunctival sac of one eye. If the pupil does not dilate in 15 minutes, 2 more drops are instilled. If the pupil dilates, a disturbance of pancreatic internal secretion is suspected.

**Disturbances of External Secretion.** Examination of the fæces may show an excess of fat (steatorrhœa). The normal dried fæces contain about 25% of fat. With pancreatic insufficiency the fat content may rise to 70% or 80%, the greater part of which is composed of neutral fat. The patient must not take any liquid paraffin by mouth for four days before the specimen of fæces is collected. Undigested muscle fibres may also be present (azotorrhœa). The urinary diastase normally measures between 6·6 and 30 units, but in some pancreatic lesions, such as acute hæmorrhagic pancreatitis, it rises to over 200 units.

**Pressure Symptoms.** A tumour of the head of the pancreas may press on the common bile duct and cause obstructive jaundice, or ascites may result from compression of the portal vein.

**Nervous Symptoms.** Acute pancreatitis may be accompanied by pain in the epigastrium or back, vomiting and shock, due possibly to irritation of the solar plexus.

## Acute Pancreatitis

*(Hæmorrhagic, Suppurative and Gangrenous Pancreatitis)*

**Etiology.** Acute pancreatitis is usually due to bacterial infection, less often to trauma. The organisms, such as the *Bacterium commune* (*B. coli*) or streptococci, gain access either from the gall-bladder, bile ducts or intestine, and ascend the pancreatic duct; less frequently the infection is blood-borne. *Predisposing causes:* 1. Age: Usually over 40 years. 2. Sex: Males predominate. 3. Nutrition: Especially the obese, or following a heavy meal. 4. Gall-stones and infection of the biliary passages. 5. Other diseases: Such as mumps, enteric fever and small-pox.

**Pathology.** The steapsinogen and trypsinogen of the pancreatic secretion become activated in the gland. This can be effected by bacteria. Further, trauma or hæmorrhage, resulting in a local autolysis of pancreatic tissue, produces an activating substance which converts trypsinogen into trypsin. The pancreas and adjacent peritoneum show varying degrees of change, such as œdema, hæmorrhage, fat necrosis or gangrene. Local suppuration may also occur. Fluid containing blood may be present in the lesser peritoneal sac or in the general peritoneal cavity. Pearly-whitish areas of fat necrosis may be seen in the pancreas,



omentum, mesentery and retro peritoneal fat. The toxæmia results from autolytic substances produced in the gland.

**Clinical Findings** The patient is suddenly taken ill with intense pain in the epigastrium and across the lower part of the back of the chest. He feels sick, vomits and collapses.

**On Examination** In a severe case the patient is desperately ill, pale, cold sweating, and at times cyanosed. The epigastrium is extremely tender and there is some, but not very marked, muscular rigidity in the epigastrium. Bluish mottling of the abdominal wall may be noted. There may be definite tenderness in the left costo-vertebral angle. The patient does not keep still as he does with a perforated gastric ulcer. *The abdomen becomes distended, the bowels are confined, but flatus is passed. No intestinal movements can be heard with the stethoscope. The temperature is usually sub-normal unless suppuration or gangrene is present. The pulse is frequent and feeble. The vomit often contains bile. The urinary diastatic index is about 200, and the urine may contain sugar and acetone bodies. Loewi's mydriatic test is positive in some cases. Mild cases also occur in which the diagnosis is established by the diastase test.*

**Differential Diagnosis** Acute pancreatitis resembles in some respects other acute abdominal emergencies, such as a perforated gastric or duodenal ulcer or intestinal obstruction. Coronary disease may be simulated in other cases. The characteristic features of acute pancreatitis are the age of the patient, the frequent history of gall bladder trouble, the absence of a history pointing to a gastric or duodenal ulcer, the cyanosis, the persistent vomiting, collapse, and, above all, the high diastatic index of the urine.

**Course and Complications** If untreated, the course is generally rapidly progressive, the patient being overwhelmed with toxæmia. Suppuration or gangrene may occur as complications, or a pancreatic pseudo-cyst may form in the lesser peritoneal sac. Diabetes mellitus has been recorded developing five years after a successful operation for acute pancreatitis.

**Prognosis** Unless an operation is rapidly performed in severe cases, death usually occurs in a day or so. Sudden death may be due to acute hæmorrhagic pancreatitis. When mumps is the cause of the disease the prognosis is good as suppuration and necrosis of the gland do not occur.

**Treatment** In mild cases rectal salines containing 5% dextrose and hypodermic injections of small doses of morphine should be given. Operation is contra-indicated. In severe cases the abdomen should be opened and the pancreas drained. The gall bladder should also be drained, if infected.

### Subacute Pancreatitis

**Etiology.** Subacute inflammation of the pancreas may result from infection associated with gall stones, ulceration of the stomach or duodenum.

**Pathology** Localised areas of pancreatic necrosis are formed

**Clinical Findings.** The patient experiences recurrent attacks of epigastric pain, which spreads to the left hypochondrium, or to the back to the left of the spine in the lower thoracic region. The pain may also be felt in the left scapular or left iliac regions.

**On Examination:** The patient during the attacks is often pale, slightly cyanosed and collapsed. There is epigastric tenderness and rigidity. The stools usually contain no excess of fat.

**Differential Diagnosis.** The pain has to be differentiated from that due to gastric or duodenal ulcer or to renal colic.

**Course and Complications.** Recurrent attacks are prone to occur.

**Prognosis.** With adequate treatment the attacks can often be prevented.

**Treatment.** The patient should be starved for three or four days—only fluids being allowed by mouth. For the next week the diet should be chiefly carbohydrate, very little fat or protein being allowed. Subsequently investigations should be made to determine whether an ulcer of the stomach or duodenum, or gall-stones, are present and, if so, adequate treatment instituted.

### Chronic Pancreatitis

**Etiology.** Chronic inflammation of the pancreas is usually due to bacterial infection of the pancreatic lymphatics, the organisms being derived from the gall-bladder or the intestine. It may also be secondary to obstruction of the pancreatic duct, as by a gall-stone in the ampulla of Vater, a pancreatic calculus, inflammations of the pancreatic ducts or carcinoma of the pancreas. Arteriosclerosis of the pancreatic vessels, syphilis or hæmochromatosis are also causative factors.

**Pathology.** In chronic interstitial pancreatitis the fibrosis may be interlobular, when it is often localised to the head of the gland, or interacinar when it is more diffuse. In chronic catarrhal pancreatitis there is inflammation of the pancreatic ducts.

**Clinical Findings.** *Chronic interstitial pancreatitis:* The patient is usually an adult who complains of epigastric discomfort or pain, or of aching in the back, flatulence and the passage of bulky, pale and pasty stools. *Chronic catarrhal pancreatitis:* The patient has periodical attacks of nausea, shivering, pain in the epigastrium and the back, vomiting and diarrhoea with large motions.

**On Examination:** Epigastric tenderness is present, the conjunctivæ may be slightly icteric, the stools contain an excess of fat (see p. 67) and undigested muscle fibres (see p. 67). The urine may contain glucose, especially in the case of interacinar pancreatitis. Further, Loewi's eye test (see p. 67) may be positive and the urinary diastatic index raised.

**Differential Diagnosis.** Chronic pancreatitis must be diagnosed from sprue and from digestive disturbances due to biliary insufficiency. In sprue the history of residence abroad, the characteristic tongue and anæmia and the excess of split fat in the faeces usually serve to establish the diagnosis. With biliary insufficiency there is also excess of split fat in the faeces and diminution of bile pigments.

**Course and Complications.** The course extends often over several years, the patient gradually losing weight. The common bile duct may be compressed in the head of the pancreas, with resultant jaundice.

**Prognosis.** This is unfavourable, as regards complete recovery.

**Treatment.** A fat poor diet should be given. Eggs should not be eaten. The following articles are permitted. Toast, thin bread and butter, minced meat, fish such as cod, hake or haddock, green vegetables, fruit, and salads. All the food should be well chewed. Sod salicyl gr 20, and sod bicarb gr 30, should be taken t.i.d. to disinfect the pancreatic ducts. Calomel gr  $\frac{1}{2}$  should be taken at night and a small dose of salts (mag sulph gr 30) in the morning. The pancreatic ducts may be further drained by passing a duodenal tube and injecting through it 40 mls of a 25% solution of mag sulph and subsequently aspirating the duodenal contents. If there is cholecystitis or gall stones cholecystectomy should be performed. The common bile duct may also require surgical drainage.

### Tumours of the Pancreas

Simple tumours and gumma are rare, an adenoma of the islets of Langerhans may give rise to *hyperinsulinism*, with symptoms of spontaneous hypoglycæmia such as faintness, sweating headache, etc (see p 630). Spontaneous hypoglycæmia may also be due to functional hyperinsulinism and hepatic disease. In the latter there is no hyperinsulinism but faulty glycogenesis in the liver. It may occur in such conditions as cholangitis, toxic hepatitis, Von Gierke's disease (see p 92) diffuse carcinomatosis and fatty degeneration of the liver. *Malignant tumours* are usually *carcinomata*, *sarcomata* are rare. Hyperinsulinism was first described in 1927 in association with a carcinoma arising in the islets of the pancreas.

### Carcinoma of the Pancreas

**Pathology.** The carcinoma is usually primary and situated at the head of the gland. Secondary deposits in the pancreas are less common, the primary growth being usually in the stomach. The gall bladder may be enlarged owing to compression of the common bile duct by the growth or by secondary glands in the portal fissure.

**Clinical Findings.** The patient is usually a male, over the age of 40. He complains of general weakness, loss of appetite, pain in the epigastrium passing through to the back, progressive jaundice and itching of the skin. In some rare cases the symptoms are those of recurrent hypoglycæmia, in others there are no signs, the patient complaining only of severe pain in the back in the lower dorsal region, or of girdle pains, which are relieved by heat and by sitting bent forward.

**On Examination.** The patient is wasted, and the skin is jaundiced to a variable degree, sometimes only slightly, but it may be dark green ("black jaundice"). The gall bladder may be palpable, but the pancreatic tumour can rarely be felt. The urine may contain bile pigment and sugar, the motions may be pale, and contain an excess of split fat.

**Differential Diagnosis.** It is not generally possible to distinguish a growth in the head of the pancreas from one in the bile ducts. The enlarged gall-bladder, emaciation and progressive jaundice are indicative of growth as opposed to chronic pancreatitis. A barium meal and fractional test meal serve to exclude carcinoma of the stomach.

**Course and Complications.** The course is rapidly progressive. The growth may erode into the stomach and cause hæmatemesis. Secondary deposits may occur in the long bones with a resultant anaemia. This is usually hypochromic, but at times megalocytic when nucleated red cells, and less often megaloblasts are present. The abdominal and mediastinal glands may also be invaded, and ascites may develop.

**Prognosis.** Death usually occurs in the course of a few months.

**Treatment.** Cholecystenterostomy may be performed to relieve the jaundice and itching, but there is no curative treatment.

### Cysts of the Pancreas

**Pathology.** The following varieties of cysts may occur: 1. The retention cyst: This has an epithelial lining, and forms as the result of obstruction of the pancreatic ducts, as by a stone or chronic pancreatitis. 2. The pseudocyst: This is formed by the lesser peritoneal sac, and results from trauma or acute pancreatitis. 3. The proliferative cyst and cyst-adenoma: These are multilocular and often found near the tail of the pancreas. They may form part of congenital polycystic disease of the kidneys and liver. 4. The hydatid cyst (see p. 714) and dermoid cyst.

**Clinical Findings.** The patient is usually over the age of 20. There may be a history of abdominal trauma. There may be no symptoms, or the patient may complain of pain in the epigastrium or between the shoulders, and of nausea, vomiting, constipation, jaundice and swelling of the abdomen and legs.

**On Examination:** The cyst may be felt as a rounded fluctuating swelling in the epigastrium. It commonly presents between the stomach and transverse colon, but may show above the stomach or below the transverse colon as indicated in the diagram (see Fig. 2). Its position can usually be identified with the help of a barium meal or enema. The cyst fluid, removed by aspiration, may contain pancreatic ferments.

**Differential Diagnosis.** The pancreatic cyst may be mistaken for

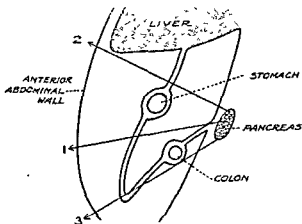


FIG. 2. DIAGRAM SHOWING POSITIONS IN WHICH A PANCREATIC CYST MAY PRESENT.

a hydatid cyst of the liver, a mesenteric cyst, a hydronephrosis, or rarely for an ovarian tumour. The anatomical relations usually suggest the correct diagnosis.

**Course and Complications** There is usually gradual enlargement of the cyst, but internal hæmorrhage causes rapid distention. Rupture results in local or general peritonitis. Malignant degeneration may occur.

**Prognosis** A complete cure, even after operation, is improbable, as recurrence often takes place. Post-operative diabetes mellitus has also been recorded.

**Treatment** An exploratory laparotomy usually permits the cyst to be drained, complete removal being generally impossible.

### Pancreatic Calculi

**Etiology** Calculi form as the result of obstruction of, or infections in, the pancreatic ducts.

**Pathology** The calculus may be composed of a mixture of calcium carbonate and phosphate, or of calcium oxalate, carbonate and cholesterol. Calculi are usually multiple, not faceted, and vary in size from a small granule to an inch in diameter. The pancreas shows chronic inflammatory changes and gall stones are often present.

**Clinical Findings** The patient is usually a male over the age of 40. There may be no symptoms, or attacks of epigastric colic and vomiting occur, the pain radiating to the region of the left scapula.

**On Examination** The patient may be tender in the epigastrium and also jaundiced. The *fæces* show azotorrhœa or steatorrhœa, and the greyish white calculi may be passed in the stools. If the calculi are rich in lime, they are shown by X ray examination.

**Differential Diagnosis** The colic resembles biliary colic, but the pain tends to radiate to the left.

**Course and Complications** Repeated attacks of colic may occur. Complications include diabetes mellitus, pancreatic abscess and peritonitis. An abscess may discharge through the abdominal wall.

**Treatment** Sedatives such as morphin sulph gr  $\frac{1}{4}$  or inhalations of chloroform are required, as for biliary colic. In some cases the calculi have been removed by operation.

## THE LIVER, GALL-BLADDER AND BILE DUCTS

**Introductory** In studying diseases of the liver, certain special methods of investigating hepatic function are available, but the results obtained are often disappointing.

The hepatic efficiency tests of most clinical value include 1 *The investigation of the pigmentary function*. This involves tests for the presence of bile in the blood, urine and *fæces*. The withdrawal and examination of bile from the duodenum by Lyon's method is not of practical value. Bile pigments in the blood are shown by the van den Bergh test, which helps to distinguish between the main types of jaundice, retention, regurgitation (obstructive) and the combined forms. Fouchet's test and the icterus index afford a less delicate indication of

the presence of bile in the blood. Bile pigments in the urine can be tested for clinically. With a deficiency of bile in the intestine the stools become fatty, there being an excess of split fat.

2. *The metabolic function.* The lœvulose and galactose tolerance tests are not sufficiently delicate to indicate fine degrees of disturbance of liver function and are now being abandoned.

3. *X-ray examination.* Cholecystography reveals the size, shape, filling capacity and contractility of the gall-bladder, and the presence of calculi. In some cases calculi are seen in a direct radiogram.

## THE LIVER

### Hepatitis

Acute inflammation of the liver occurs in association with hepatic abscess, secondary syphilis, cloudy swelling, acute yellow atrophy of the liver and toxins and infections affecting the liver. Cirrhosis of the liver may be regarded as a variety of chronic hepatitis.

### Malformations and Displacements

The malformation which is of the greatest importance clinically is the Riedel's lobe. This is a tongue-like downward projection from the right lobe of the liver, which may be mistaken for an enlarged gall-bladder, or for the right kidney. It usually occurs in women.

The liver may be displaced downwards, as by a subphrenic abscess, or upwards by an abdominal tumour or ascites. Hepatoptosis is a term applied to prolapse of the liver. It is more common in women and may give rise to abdominal dragging or to pain or biliary colic.

*On Examination:* An abdominal tumour is felt which can be pushed back into the space normally occupied by the liver.

### Hyperæmia

*Active Hyperæmia.* This may be associated with a chill, especially in tropical countries, in individuals who have suffered from malaria or dysentery. It may also result from over-eating, associated with a sedentary life and chronic constipation. It is often known as a "chill on the liver."

*Clinical Findings.* The patient complains of headache, nausea, constipation, and a sense of fulness or pain in the region of the liver.

*On Examination:* The tongue is furred, and the liver may be just palpable and somewhat tender.

*Treatment.* The patient should be kept in bed for a few days, on a milk diet, and aperients given such as calomel gr. 2 nocte, with mag. sulph. gr. 60 to 120 mane. Hot applications to the liver area are comforting. Subsequently a belt should be worn over the upper abdomen.

*Passive Hyperæmia.* This usually results from back pressure due to heart failure. There is congestion and anoxia of the central zone of the liver lobules, producing the "nut-meg" liver.

**Clinical Findings.** The patient may complain of pain in the liver region

**On Examination** In addition to the evidence of cardiac disease, the liver is enlarged and expansile pulsation may be detected on bimanual palpation, especially if tricuspid regurgitation is present. There may also be jaundice

**Treatment.** The underlying cardiac lesion must be treated. In addition, venesection or the application of three or four leeches over the liver helps to relieve the congestion

### Infarction

The intralobular branches of the portal vein may be blocked by an embolus or by thrombosis. An embolus may also occur in the hepatic artery. Thrombosis may develop in the larger branches of the portal or hepatic veins. The embolus may consist of blood clot or of a portion of new growth. The infarct is hæmorrhagic, unless due to obstruction of the hepatic artery, when it is anæmic. It is generally deep seated and gives rise to no symptoms during life, unless there is pain from an associated perihepatitis

### Perihepatitis

**Definition.** Inflammation of the capsule of the liver. This may be acute or chronic

#### Acute Perihepatitis

**Etiology** This may be secondary to a liver abscess, gumma, hydatid cyst, cholangitis etc., or it may form part of a general or local acute peritonitis

**Clinical Findings.** The patient complains of pain in the region of the liver, near the angle of the right scapula, or at the tip of the right shoulder

**On Examination** There is diminished movement on the affected side of the chest, the liver may be tender on palpation and a localised friction rub can be felt or heard

**Treatment.** The pain is best relieved by immobilising the side of the chest by strapping

#### Chronic Perihepatitis

*This may be local or diffuse.*

**Local Perihepatitis** This may be due to local affections of the liver, such as a gumma or cyst, or occur in association with passive hyperæmia due to heart disease, or adjacent to an inflamed gall bladder. It may also form part of a tuberculous or malignant peritonitis

**Clinical Findings** There are usually no symptoms, but localised pain may occur

**Diffuse Perihepatitis (Sugar iced Liver)** This is associated with chronic proliferative peritonitis (see p 108). A thick white fibrous coat forms over the liver, which can be stripped off. There is often thickening of the omentum, ascites, chronic pericarditis,

arteriosclerosis and chronic nephritis (Pick's disease). There are usually no toxæmic symptoms and no jaundice.

**Treatment.** The ascites can be relieved by aspiration, and pot. iod. should be given by mouth in doses of gr. 5 to 10 t.i.d.

### Liver Abscess

**Definition.** Suppuration in the liver.

**Etiology.** The abscess may be due to the following causes :

1. Amœbiasis (see p. 687). It is then usually single. 2. Portal pyæmia. The infection may arise in the appendix ("appendicular liver"), or in other sites such as the prostate or rectum. It may be secondary to suppurative pylephlebitis (see below). The abscesses are usually multiple. 3. Arterial pyæmia. This is often secondary to otitis media, the infection being carried to the liver by the hepatic artery. 4. Suppurative cholangitis. The infection spreads up the bile ducts. 5. Trauma. An abscess may form secondary to a wound of the liver. 6. Retrograde infection by the hepatic veins. This rarely occurs. 7. Direct spread of infection. This may be due to a subphrenic abscess or empyema of the gall-bladder. 8. Suppuration in a hydatid cyst. 9. Actinomycosis.

**Clinical Findings.** The clinical picture in amœbiasis and hydatid infection is considered separately (see pp. 687, 714). With pyæmic abscesses the patient is very ill, with a swinging temperature and rigors. The liver is usually enlarged and tender, and there may be some icterus.

**Prognosis.** With multiple abscesses this is usually hopeless.

**Treatment.** No special treatment is available.

### Suppurative Pylephlebitis

**Definition.** This term is synonymous with portal pyæmia (see p. 577). Suppuration occurs in the tributaries of the portal vein, in the vein itself and its branches.

**Etiology.** Suppurative pylephlebitis is most commonly due to an appendix abscess. Less often the condition is secondary to an amœbic liver abscess, to pus in the gall-bladder or bile ducts, to infection after rectal operations or pelvic operations in women.

**Pathology.** The liver is enlarged, perihepatitis is usually present, and abscesses may be seen on the surface or only on section of the organ. The portal vein or its branches contain disintegrating blood clot with a variable amount of pus.

**Clinical Findings.** The disease usually begins during convalescence from an operation for an appendix abscess. The patient becomes gravely ill with rigors, sweating and increase of temperature and pulse rate. This is followed in a few days by pain in the region of the liver.

**On Examination :** The patient looks ill, the tongue is dry and furred, and there may be slight icterus. The liver is found to be enlarged and tender. The spleen is not usually enlarged. The blood : There is usually a leucocytosis, but the systemic blood culture is often sterile. The temperature is irregular; it may be remittent or intermittent.

**Differential Diagnosis.** The increase in the gravity of the patient's



condition with the rigors and hectic temperature suggest pyæmia, and the evidence of liver involvement renders the diagnosis of portal pyæmia clear. In many cases however, it is difficult, if not impossible, to diagnose during life. There may be no definite localising signs pointing to the primary source of the infection.

**Course and Complications** The course is progressive. Complications such as empyema or a lung abscess, may occur.

**Prognosis** Death usually occurs in one to six weeks.

**Treatment** It is doubtful whether there is any curative treatment available in established cases. Treatment is therefore palliative for relief of pain and discomfort.

### Portal Thrombosis

(*Pylethrombosis Pylephlebitis Adhæsiva*)

**Definition** Thrombosis of the portal vein.

**Etiology** Portal thrombosis is most often associated with cirrhosis of the liver, or with new growths in the pancreas, stomach or liver. In other cases it is due to syphilis of the portal vein, trauma, or to infections in the portal area such as the intestines, appendix, spleen, pancreas or gall bladder. It may occur in erythræmia vera and in splenic anæmia.

**Pathology** The wall of the portal vein or its branches is generally thickened and the blood is clotted. The spleen is usually enlarged, but the liver may be normal in size. The mesenteric veins alone may be thrombosed, with secondary gangrene of the intestine, usually in the jejunum.

**Clinical Findings** There may be hæmatemesis, acute abdominal pain or swelling of the abdomen due to ascites. With a mesenteric thrombosis (see p. 112) the symptoms resemble those of an acute abdominal emergency.

**On Examination** The spleen may be enlarged and dilated veins seen around the umbilicus.

**Differential Diagnosis** It is very difficult to make a certain diagnosis in cases of portal thrombosis, especially as it is so often associated with other severe diseases. The sudden onset of abdominal pain, hæmatemesis or ascites is very suggestive.

**Course and Complications** The thrombosis may persist for many years or rapidly prove fatal. Recurrent attacks of hæmatemesis may occur.

**Prognosis** This is very grave, but in some cases the patient survives the attack for several years.

**Treatment** If the patient survives the immediate shock, the Wassermann reaction should be determined, and if positive a course of pot iod gr 10 to 30 with liq hydrarg perchlor m 30 given t.i.d. for 2 to 3 months.

### Cysts of the Liver

The following varieties of cyst may occur: 1. Blood and degeneration. 2. Dermoid. 3. Lymphatic. 4. Endothelial. 5. Due to bile

duct obstruction. 6. Cystadenoma. 7. Hydatid. 8. Polycystic. This forms part of congenital polycystic disease of the kidneys (see p. 480). The solitary non-parasitic cyst is probably a cystadenoma arising from a congenital aberrant bile duct.

**Clinical Findings.** Unless the cyst is large it cannot be felt. In polycystic disease, however, the enlarged and irregular surface of the liver can often be detected. Cysts may rupture into the peritoneum, causing severe shock, or a fatal hæmorrhage may take place into the cyst. A cyst may press on the bile ducts or duodenum, suppuration may occur, or the pedicle become twisted.

### Hydatid Cysts

**Etiology.** The disease is due to infection with the *Echinococcus granulosus* (*T. echinococcus*) (see p. 714).

**Pathology.** The cysts may be multiple or single, deeply embedded in the liver substance or projecting from the surface. The liver is the organ most commonly affected in hydatid disease. The structure and contents of the cyst and the changes they may undergo are described on pp. 714, 715.

**Clinical Findings.** If the cyst is small or deep-seated, it may cause no symptoms. In other cases the patient complains of aching or of pain in the region of the liver, and he may give a history of residence in an infected area.

**On Examination:** The liver may be enlarged and a rounded swelling felt, depending upon the position of the cyst. A hydatid thrill (see p. 714) is rarely palpable. The blood may show an eosinophilia. The Casoni intradermal test is largely used in Australia. It consists in the intradermal injection of 0.2 mil. of filtered hydatid fluid, obtained from a cyst of a sheep. A positive reaction is shown by the formation of an urticarial wheal in about 10 minutes, followed in a few hours by erythema and œdema around the site of the injection. There may also be a febrile reaction.

**Differential Diagnosis.** A hydatid cyst, if palpable, must be distinguished from a gumma or carcinoma of the liver, or an enlarged gall-bladder. It may also simulate a pleural effusion, if situated posteriorly. The diagnosis is usually suggested by the history of the case, the negative Wassermann and the positive blood and intradermal tests.

**Course and Complications.** The cyst may cure itself by inspissation. On the other hand, it may continue to grow, it may suppurate or a hæmorrhage may occur into it. Rupture may take place into the abdomen, stomach, intestine, etc.

**Prognosis.** The mortality rate is about 15%.

**Treatment.** The cyst should not be tapped. If causing symptoms, it should be removed as completely as possible by an open operation. Aspiration is dangerous, as severe shock or death may occur if any of the fluid enters the peritoneum.

### Cirrhosis of the Liver

**Definition.** Hardening of the liver, due to the formation of fibrous tissue. The varieties of cirrhosis may be classified as follows: 1.

Portal (multilobular)    2 Biliary (unilobular)    3 Pericellular    4  
 Capsular    5 Pigmentary    6 Mixed types

### Portal Cirrhosis

(Multilobular    Alcoholic    Lænnec's Atrophic Cirrhosis  
 Hob nail Liver    Gin drinker's Liver)

**Etiology.** An irritant is conveyed to the liver in the portal vein. The nature of the irritant is not certain in every case, but alcohol is usually considered to be a factor of some importance. Cirrhosis may, however, occur apart from alcohol, as in young children, in races such as the Hindoos, who take no alcohol but eat spices such as ginger and cardamon, and in animals, such as the butcher's cat. Organisms of low virulence may settle in the liver, such as the *Bacterium commune* (*B. coli*) and cause a condition of sub infection disintegrating there and liberating endotoxins. Chronic gastritis is also a factor, chemical irritants may be absorbed from the stomach or intestine, races indulging in highly spiced foods being liable to cirrhosis. It may also occur in Wilson's disease (see p. 341) and in Banti's disease (see p. 510). **Pre disposing causes** 1 Age. Usually over 40, but cirrhosis may occur in children. 2 Sex. Males predominate. 3 Occupation. Especially those working in the liquor trade and commercial travellers. 4 Climate and locality. It is more common in temperate zones.

**Pathology.** Typically the liver is small (Lænnec's atrophic cirrhosis). It may be enlarged owing to fatty changes. The surface is irregular, the projections being called 'hob nails'. These are formed by hyperplasia of liver cells, and are yellowish on section (*Kippos* = yellow). The liver is tough on section owing to the bands of fibrous tissue which surround several lobules. The spleen may be enlarged. A compensatory venous circulation is established on account of the portal obstruction. The main channels opened up are Veins running from the bare area of the liver to the phrenic and intercostal veins. An anastomosis between the œsophageal veins and the coronary veins of the stomach. Dilated veins are often present in the lower part of the œsophagus, and in the stomach there may be erosions of the mucous membrane or dilated veins. Enlargement of the para umbilical vein of Sappey, running in the falciform ligament and connecting the epigastric and portal veins. Dilatation of the veins around the umbilicus gives rise to the appearance known as the *caput Medusæ*. An anastomosis between the inferior mesenteric and the hæmorrhoidal veins. Hæmorrhoids are not common. Enlargement of the retro-peritoneal veins of Retzius, establishing communication between the portal tributaries and those of the inferior vena cava.

**Clinical Findings.** The patient is usually an adult male, over the age of 40, who gives a history of over indulgence in alcohol for several years. In the early stages the symptoms are due to chronic gastritis. The patient complains of flatulence, nausea, retching or vomiting small quantities of fluid on rising, and of a lack of desire for breakfast. He may have headache, recurrent attacks of epistaxis and constipation. The first symptom in some cases is a hæmatemesis of half a pint or more. In

the later stages, the patient complains of increasing weakness, dyspnoea, and perhaps of swelling of the abdomen and legs.

*On Examination:* The facies is usually suggestive, the patient being well nourished with dilated venules on the cheeks, and the eyes are congested and watery. The tongue is furred and the breath is often offensive. The liver may be felt enlarged, a little irregular and firm, and the spleen may be just palpable.

Later, the patient presents the typical hepatic facies; the face is thin and the nose prominent; the complexion is rather sallow, and dilated venules are seen on the cheeks and nose. The chest, arms and legs are wasted, but there may be swelling of the legs due to œdema. The abdomen is distended. Free fluid may be detected in the abdomen (see p. 111), and the liver may be felt on "dipping" below the costal margin to be firm and irregular. In other cases the liver is not palpable until the fluid has been removed from the abdomen, or the liver may have shrunk and not be palpable. The dilated veins may be seen in the abdominal wall, as described above. Symptoms of toxæmia (cholæmia) may be present; the patient complains of headache, inability to concentrate, and drowsiness. The skin is usually sallow or slightly icteric, and muscular tremors or twitching may be seen. The urine is concentrated and may contain bile. The blood shows an excess of bile pigment giving the biphasic or delayed van den Bergh reaction, and in the later stages of the disease a macrocytic anæmia may develop. The temperature is often irregular and slightly raised.

*Differential Diagnosis.* The symptoms in the early stages are those of chronic gastritis, and there may be no indication that the liver is cirrhotic until pressure manifestations occur, such as hæmatemesis, or until the liver is definitely enlarged. Other causes of hæmatemesis, such as gastric ulcer, must then be excluded. The barium meal and test meal will afford evidence of gastric ulceration.

Enlargement of the liver necessitates a consideration of other causes than cirrhosis, such as malignant disease, syphilis, splenic anæmia, passive hyperæmia, amyloid degeneration, a hydatid cyst, an abscess, leukæmia or Hodgkin's disease. In malignant disease (see p. 90) the liver is usually more irregular, definite nodules with central umbilication are palpable in some cases, and jaundice tends to be more intense. In some cases, however, the liver is infiltrated with secondary deposits, and no jaundice is seen. The growth is usually secondary to a lesion elsewhere. In syphilis the Wassermann reaction is positive, and there is amelioration of the condition with the administration of iodides. In splenic anæmia (see p. 510) the spleen is usually considerably enlarged before the liver enlarges. In passive hyperæmia (see p. 73) there is evidence of heart failure, and expansile pulsation may be detected in the liver. In amyloid degeneration the patient is usually suffering from a definite wasting or suppurative disease. The blood examination is of value in the diagnosis of hydatid disease, abscess and leukæmia. In Hodgkin's disease, enlarged glands are usually found elsewhere. The alcoholic history and appearance of the patient are also of value.

In the ascitic stages other causes of ascites must be considered (see

p 110) Cholæmia resembles uræmia in some respects clinically, and the non protein nitrogen content of the blood may be raised

**Course and Complications** In the early stages the disease may pursue a very slow course and arrest may even occur When ascites has developed the end is not far distant A patient with ascites due to portal cirrhosis rarely lives to be aspirated more than once or twice Complications include Portal thrombosis, pulmonary tuberculosis, tuberculous peritonitis, hæmorrhagic pleural effusion, pneumonia, and peripheral neuritis

**Prognosis** This is very grave, except in the early stages and the patient usually dies a few years after the diagnosis is made Cholæmia is a fatal complication

**Treatment** In the early stages the patient must give up all alcohol and spiced foods, and no tinctures or spirits must be given as medicines The diet should be largely lacto vegetarian The bowels should be kept acting daily with salines such as mag sulph gr 60 mane If the Wassermann reaction is positive a course of pot iod gr 5 to 60 t i d should be given for 2 to 3 months If there is hæmatemesis no morphine must be given as owing to failure of hepatic detoxication even gr  $\frac{1}{2}$  may prove fatal For ascites the fluid intake should be restricted to 2 pints in the 24 hours and diuretics such as Guy's pill (pil digital co B P C) should be given 1 pill t i d or theophyll et sod acetat gr 2 t i d for 3 days or Salyrgan (mersalylum B P) or Novurit can be administered as described on p 228 If there is much abdominal discomfort from the ascites the fluid should be slowly drained off The puncture should be made with Novocain (procain hydrochlor B P) anæsthesia in the mid line half way between the umbilicus and symphysis pubis after the bladder has been emptied If the fluid is loculated, it can sometimes be drained off by a puncture in the flank For the cholæmic symptoms rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours, or an intravenous drip of 1 to 2 pints of normal saline containing 5% dextrose

### Biliary Cirrhosis

#### (Unilobular Cirrhosis)

There are two types which will be described separately Hanot's cirrhosis and obstructive biliary cirrhosis

### Hanot's Cirrhosis

#### (Hypertrophic Cirrhosis with Chronic Jaundice)

**Definition** A disease of young people characterised by enlargement of the liver, persistent jaundice, and periodical febrile attacks

**Etiology** The cause is not known It may result from infective cholangitis. Alcohol is probably not a causative factor **Predisposing causes** 1 Age 20 to 30 years, but it may occur in children 2 Sex Equal incidence or slightly more frequent in males 3 Infections Such as enteric fever

**Pathology** The infection is believed to be blood borne in the

majority of cases. Post-mortem the liver is enlarged, with a smooth surface and dark green colour. It is firm on section, and a fine perilobular fibrosis is present; in the late stages the fibrosis may be multilobular. New-formed "pseudobile canaliculi" may be seen in the fibrous tissue, due to a hyperplasia of bile-duct cells. The spleen is normal or enlarged.

**Clinical Findings.** The disease is very rare, and some authorities consider it to be non-existent. This opinion, however, is probably incorrect. The onset is gradual, the patient complaining of loss of appetite, attacks of nausea or diarrhoea, jaundice and itching of the skin. Pain may be felt in the region of the liver or of the spleen.

**On Examination:** The patient is jaundiced, but not usually very deeply. The liver is found to be enlarged, and may extend from the fourth rib to 4 inches below the costal margin. It feels smooth and firm. The spleen may be palpable. There is no ascites. The fingers are often clubbed, and hypertrophic osteo-arthropathy may be seen in the wrists or other joints. The blood: There is a hæmolytic anæmia, but the leucocytes are often increased up to 12,000 per c.mm. or more. The van den Bergh reaction is indirect. The urine: This contains urobilin. The fæces: Urobilin is present.

**Differential Diagnosis.** Hanot's cirrhosis may be confused with acholuric jaundice, Banti's disease, portal cirrhosis, and catarrhal jaundice. The characteristic features of Hanot's cirrhosis are the persistent jaundice of a rather mild degree, the absence of ascites, and the enlarged liver. The red cells show no increased fragility as they do in acholuric jaundice. In Banti's disease there is usually a leucopenia, and hæmorrhages occur before the onset of jaundice. Portal cirrhosis is characterised by a milder degree of jaundice and the tendency to ascites. In regurgitation jaundice the fæces contain no bile.

**Course and Complications.** The course is usually slowly progressive, but there is a tendency to periodical febrile attacks with intensification of the jaundice. Complications include intercurrent diseases, such as erysipelas or pneumonia. Cholæmia occurs in the final stages, and occasionally there is ascites or gastric hæmorrhage.

**Prognosis.** The disease is usually fatal in about 5 or 6 years from its onset.

**Treatment.** This is largely symptomatic. The diet should be mainly lacto-vegetarian, with some fish, but little meat. No alcohol should be allowed. The bowels should be kept open daily with a morning dose of mag. sulph. gr. 60, and calomel gr. 2 given once a week at night.

### Obstructive Biliary Cirrhosis

#### (Charcot's Cirrhosis)

**Etiology.** Charcot's cirrhosis results from obstruction of the bile ducts, as by a calculus, the bile acting as an irritant. There is possibly an associated blood-borne infection of micro-organisms or toxins.

**Pathology.** At autopsy, the liver is usually contracted. The

surface is irregular and the colour is dark green. On section it presents a honeycomb appearance due to dilatation of the bile ducts. Fibrosis is present around the bile ducts, and thus fibrosis may be unilobular or multilobular. Calculi may be present in the extrahepatic bile ducts. The spleen is not usually enlarged.

**Clinical Findings.** The onset is gradual, with progressive jaundice.

**On Examination.** The liver is not generally palpable, but an enlarged gall bladder may be felt. The blood. The van den Bergh reaction is direct. The urine. Bile pigment is present. The fæces are pale owing to absence of bile.

**Differential Diagnosis.** The diagnosis involves consideration of the causes of regurgitation (obstructive) jaundice, especially that due to gall stones. The onset of cholæmic symptoms indicates that there is further failure of hepatic function.

**Course and Complications.** The course is progressive, unless the obstruction is removed before irreparable damage has been done to the liver.

**Prognosis.** Early treatment may effect a cure. The outlook is hopeless with the onset of cholæmia.

**Treatment.** The obstruction should be removed, if possible, by operation.

### Pericellular Cirrhosis

This is met with in congenital syphilis, and at times in miliary tuberculosis. The liver is enlarged, yellow and tough.

### Capsular Cirrhosis

Cirrhosis of the liver may be secondary to chronic perihepatitis. The cirrhosis is not of clinical importance, apart from the perihepatitis.

### Pigmentary Cirrhosis

This includes hæmochromatosis (see p 645). Further, in anthracosis coal dust may be deposited in the liver, and in malaria, pigment derived from the blood may be present in the liver, the cirrhosis being unilobular or multilobular.

### Mixed Types of Cirrhosis

In portal cirrhosis and hypertrophic biliary cirrhosis, the cirrhosis may be multilobular, unilobular or pericellular.

### Jaundice

#### (Icterus)

**Definition.** A condition characterised by excess of bile pigment in the blood, with a yellow discoloration of the skin and conjunctivæ.

**Physiology and Pathology.** Bile pigment is made from red cells engulfed by the phagocytic reticulo-endothelial cells in the bone marrow, spleen and liver. The reticulo-endothelial cells in the liver (Kupffer cells) are situated in the walls of the intrahepatic branches of the portal vein. The bilirubin is removed from the blood in the liver by the epithelial (polygonal) cells, and excreted *via* the bile canaliculi.

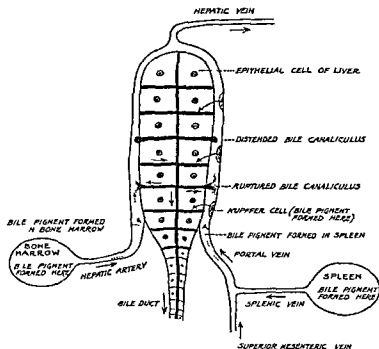


FIG 3 DIAGRAM (MODIFIED FROM RICH) SHOWING PART OF A LIVER LOBULE.

If an excess of bile pigment is brought to the liver by the portal vein and hepatic artery, and the liver cell function is simultaneously depressed, excess of bilirubin leaves the liver by the hepatic vein and there is retention jaundice. If the liver cells are necrosed or the bile ducts are obstructed, bile pigments and salts regurgitate into the radicles of the hepatic vein and regurgitation jaundice results.



to the bile duct. In the intestines the bilirubin is converted by the action of micro-organisms into urobilinogen and urobilin (stercobilin). These bodies are reabsorbed and again excreted by the liver as bilirubin and biliverdin. Urobilinogen is colourless and is normally excreted in the urine, but urobilin only appears in minute traces in the urine. In certain diseases, such as acholuric jaundice, in which the liver cells are presumably damaged, the liver fails to recover the urobilin, and urobilinuria results.

*The Classification of Jaundice.* None of the classifications propounded can be accepted with enthusiasm, and this is inevitable owing to the inadequacy of present knowledge. The old division of jaundice into obstructive and non-obstructive groups has the merit of being non-committal as regards the nature of the second group. McNee's classification into obstructive, toxic and infectious hepatic, and hæmolytic cases subdivides the non-obstructive group, but is liable to obvious criticism. The nomenclature is mixed, being based both on pathogenesis and on etiology. Further, special cases of jaundice could equally well be placed in two groups. Arnold Rich has proposed a classification based on clinical laboratory findings and theoretical views as to pathogenesis. He recognises two main types of jaundice: 1. *Retention jaundice.* In these cases there is excessive production of bile pigments by the reticulo-endothelial cells, and diminished excretion of bile by the liver cells. The latter is due to subnormal function caused by various factors such as anoxæmia, febrile disease or immaturity of liver cells. The excess of bile pigment does not pass through the epithelial cells of the liver and enters the general circulation by the hepatic vein (see Fig. 3). The causes of this group are given in Table I. (see p. 85). 2. *Regurgitation (obstructive) jaundice.* The bilirubin in the blood is not excreted normally, either owing to necrosis of the liver cells or to obstruction in the bile canaliculi or ducts. The ducts then rupture (see Fig. 3) and bile passes back into the blood channels of the liver. The causes of this type are detailed in Table II. (see p. 85). *A combined form of jaundice* may also occur, in which there is an excessive production of bile pigments and a regurgitation of bile which has been excreted into the canaliculi.

The van den Bergh test is invoked to distinguish these types of jaundice. This test is assumed to show the amount and variety of bile present in the blood, according to the reaction given. *The direct reaction:* A purple colour develops immediately the diazo reagent is added to the serum. This indicates regurgitation (obstructive) jaundice. *The indirect reaction:* No colour change occurs on adding the diazo reagent to the serum, but after standing a red colour may or may not develop. If, however, alcohol is added to the serum before the diazo reagent an immediate colour change occurs. This indicates retention jaundice. The explanation of the difference in the reaction given is as follows: The indirect reaction results when bilirubin alone is present in excess in the blood, the direct reaction when all the constituents of bile, i.e., pigment, salts and cholesterol, are present in excess. It seems that, when bile pigment is present alone, it is adsorbed to the plasma proteins, and so

the colour change is not readily obtained, whereas the presence of the other constituents of bile prevent this adsorption from occurring and the bile pigment gives an immediate direct reaction *The biphasic reaction* In some instances a slight but immediate, reddish colour change takes place when the reagent is added to the serum This deepens to violet on standing for half an hour or longer The biphasic reaction indicates a combined type of jaundice The van den Bergh test definitely allows the amount of bile pigment present in the blood to be estimated This is done in terms of units 1 unit being equivalent to a concentration of bile in the serum of 1 in 200 000 Normally blood contains between 0.2 and 0.5 van den Bergh units of bile pigment or 0.2 to 1.7 mg per 100 c.c. Before the bile pigment appears in the urine the blood concentration must reach 4 units *Latent jaundice* is a condition in which there is an excess of bilirubin in the blood usually up to 4 units, but no jaundice is found clinically It occurs in pernicious anaemia *Dissociated jaundice* An excess of bile salts may accumulate in the blood, without there being a rise in the bilirubin content This may cause pruritus and bradycardia in certain cases of cirrhosis hepatis Retention of bile pigment, with a normal excretion of the bile salts is found in the later stages of catarrhal jaundice The blood phosphatase (see p. 619) has been found to be considerably raised in cases of marked obstructive jaundice, whereas it is normal in other types of jaundice

The causes of the various types of jaundice are detailed in Tables I and II (see below) which are those given by Arnold Rich (*Johns Hopkins Hosp. Bull.* 1930 47, 338)

Rich's view has the merit of introducing the theory that excessive production of bile pigment alone, as in a hæmolytic jaundice, will not cause jaundice An additional factor is required, derangement of the liver cells interfering with excretion The van den Bergh test, however, is of little practical value in determining the type of jaundice present If there is much bile pigment in the blood, a direct reaction is obtained, if only little bile pigment then the reaction is delayed Further, Rich's distinction between liver cells which are the subject of cloudy swelling and those which are necrosed is a subtle one The former result in retention jaundice and the latter in regurgitation jaundice Totally dissimilar clinical types of jaundice, such as a toxic process caused by chloroform or phosphorus and an obstructive process due to a calculus, are classed together by Rich as regurgitation jaundice

### Retention Jaundice

### Hæmolytic Jaundice

**Etiology** This may occur in acholuric jaundice (see p. 495) in splenic anaemia (see p. 510) in pernicious anaemia (see p. 487), and in paroxysmal hæmoglobinuria (see p. 444) It may also result from blood transfusion when the blood is incompatible, or in association with *Diphyllobothrium latum* infestation (see p. 713)

**Pathology** The spleen is often enlarged, and there is anaemia In

TABLE I.

<b>I. Retention jaundice.</b> Laboratory tests : Blood : ++ indirect v. d. Bergh. Stools : ++ urobilin. Urine : ++ urobilin. Cause : Over-production of bilirubin + subnormal liver function caused by	A. Anæmia caused by	1. Anæmia.	Pernicious anæmia. Hemolytic jaundice. Sick-cell anæmia. Paroxysmal hæmo- globinuria. Mismatched trans- fusion. Phenylhydrazine poisoning.
		2. Chronic pas- sive conges- tion.	Cardiac decompensa- tion (especially with pulmonary infarction).
	B. Febrile disease asso- ciated with anoxæmia re- sulting from	1. Anæmia.	Hæmolytic septi- cæmias.
		2. Pulmonary consolidation.	Malaria. Black-water fever. Lobar pneumonia.
	C. Immaturity of liver cells in	Newborn.	Icterus neonatorum.
	D. Undeter- mined.		Hanot's cirrhosis.

TABLE II.

<b>II. Regurgita- tion Jaundice.</b> Laboratory tests : Blood : .+ + direct v. d. Bergh. Stools : de- creased uro- bilin. Urine : + bili- rubin and bile salts. Cause : Rupture of canaliculi, caused by	A. Necrosis of liver cells, caused by	1. Toxic agents.	(a) Chemical.	Chloroform. Carbon tetrachloride. Phosphorus. Salvarsan. etc.
			(b) Vegetable.	Mushroom poisoning.
			(c) Bacterial.	Yellow fever. Congenital syphilis. Weil's disease.
			(d) Undeter- mined.	"Idiopathic" acute yellow atrophy. Lænnec's cirrhosis.
	B. Obstruc- tion of bile ducts, caused by	2. Severe degrees of A. and B.		Table I.
			(a) Calculi.	Cholelithiasis.
			(b) Inflamma- tory exudate	Pancreatic calculi. Cholangitis.
			(c) Parasites.	Fasciola hepatica. Ascaris. etc.
	C. Undeter- mined.	1. Plugging.	(d) Neoplasms.	Tumours of bile ducts.
			(a) Scarring.	Chronic cholangitis. Syphilitic cirrhosis.
			(b) Malforma- tions.	Congenital stenosis or atresia of ducts.
			(c) Neoplasms.	Primary or secondary tumours involving bile ducts.
		2. Stricture.	(a) Inflamma- tory masses in liver.	Abscess. Gumma. Tubercle. Hodgkin's disease.
			(b) Parasitic masses.	Echinococcus cyst.
			(c) Vascular tumours.	Aneurysm of hepatic artery.
			(d) Inflamma- tory tissue outside liver.	Peritoneal adhesions.
		3. Pressure.	(e) Neoplasms.	Tumours of pancreas, gall-bladder, liver, etc.
			(f) Enlarged hepatic lymph nodes	Tumour metastases. Hodgkin's disease. Tuberculosis.
				Catarrhal jaundice.

these cases the renal threshold for bilirubin is often raised, so that the blood bilirubin is over 4 units, without bilirubin appearing in the urine. The jaundice is usually slight.

### Jaundice in New-born Infants

#### (*Icterus Neonatorum*)

The following varieties are described —

*Mild Types.* Physiological, appearing during the second or third day of life and lasting for 1 or 2 weeks, and catarrhal jaundice. The former is probably due to an increased formation of bile pigment, dependent upon polycythæmia, and a diminished excretory power of bile pigment by the liver cells, as shown by dye excretion tests.

*Severe Types.* These may be due to congenital stenosis or absence of the bile duct, gall stones, or congenital syphilis of the liver. These cases conform to the regurgitation type of jaundice. Infections of the umbilicus or in the intestines may cause severe *icterus neonatorum*. Two types of infective jaundice are described, that of Winckel, in which there may be hæmaturia, and that of Buhl, which may show hæmorrhage from the stomach intestines or umbilicus. *Icterus gravis neonatorum with erythroblastosis* is a severe and often familial type of jaundice. There is anæmia and an abnormally large number of nucleated red cells in the blood. The liver and spleen are enlarged and hæmatopoiesis occurs to an abnormal degree in these organs. Jaundice is present at birth or appears within 24 hours. Oedema may be present and purpura is not uncommon. Some infants recover spontaneously, and some who survive develop subsequently nervous lesions due to jaundice of the striato pallidal portions of the brain (*kernicterus*). The manifestations of this include choreo athetosis, extrapyramidal spasticity, opisthotonos and mental deficiency. Treatment consists in the immediate intramuscular injections of 5 to 10 mls of whole blood derived from the father. If there is no improvement within a few hours, as shown by the general condition of the baby, the jaundice, the number of red cells and percentage of hæmoglobin, 60 mls of group O blood should be injected intravenously and repeated next day. For the hypoprothrombinæmia vitamin K, 1 mil of Kapon is injected intramuscularly every other day. If the condition is still deteriorating, immediate splenectomy affords a hope of cure.

### Regurgitation Jaundice

#### (*Obstructive Jaundice*)

The obstruction may be extrahepatic or intrahepatic.

*Extrahepatic Obstruction Causes in the bile duct.* These include a gall stone, a round worm, a hydatid cyst or a projection of a carcinoma arising in the gall-bladder. *Causes in the wall of the duct.* A new growth, stricture or catarrhal inflammation. *External pressure on the duct.* Carcinoma of the head of the pancreas, chronic pancreatitis, enlarged glands or a gumma in the portal fissure, carcinoma or ulcer of the stomach or duodenum, renal or suprarenal tumours, an aneurysm of the celiac axis, hepatic or mesenteric arteries.

**Intrahepatic Obstruction.** This may be due to cholangitis, cirrhosis or carcinoma of the liver or to necrosis of the polygonal cells of the liver.

The varieties of obstructive jaundice, such as that due to carcinoma of the liver or pancreas, or to gall-stones, etc., are considered under their respective headings.

### Catarrhal Jaundice

**Etiology.** No causative organism has been isolated. *Predisposing causes:* Age: Young adults. Exposure to chill, and possibly strain and overwork.

**Pathology.** The disease is rarely fatal, so that the morbid anatomy is doubtful. There may be inflammation of the duodenum, the extra- and intra-hepatic bile ducts and of the hepatic polygonal cells.

**Clinical Findings.** The patient is usually a young adult, who complains of vague discomfort or pain in the epigastrium, nausea and general depression for a few days. He may also notice that the skin itches, the urine is dark brown in colour, and the motions are pale. The icterus first appears in the eyes, and the skin is subsequently affected. The patient rarely complains of xanthopsia (yellow vision).

**On Examination:** The whites of the eyes are yellow, and the skin has usually a canary yellow colour. This cannot be seen in artificial light. The tongue is furred and the temperature is often raised to 99° or 100° F. The pulse is rather slow. There is usually tenderness over the liver or gall-bladder, and they may be palpable. The urine is mahogany coloured and contains bile pigment. Bile salts are usually only present for the first two days of the illness. Urobilin may be present at the beginning and end of the illness. The motions are rather offensive and large, pale or putty coloured. They contain an excess of split fat and a diminished amount of urobilin. The blood in the early stages gives a direct van den Bergh reaction, but in the later stages the reaction may be biphasic. Bile pigment is not usually present in such body fluids as the saliva, tears, sweat, milk or cerebro-spinal fluid.

**Differential Diagnosis.** The obstructive nature of the jaundice is indicated clinically by the presence of bile pigments in the urine and their reduction in the stools. This is confirmed by the van den Bergh test. The age of the patient suggests the jaundice is catarrhal. Some cases of Weil's disease (see p. 698) and of glandular fever (see p. 587) have been mistaken for catarrhal jaundice. If the jaundice persists for several weeks, the possibility that it is due to calculus, or to Hanot's cirrhosis will arise, and in older persons it may be mistaken for carcinoma of the liver, bile passages or pancreas. With complete obstruction of the bile ducts, no bilirubin enters the intestine and so no urobilinogen or urobilin is present in the urine. If the galactose tolerance test shows a marked rise in the blood sugar, the jaundice is probably catarrhal in origin, and not due to a calculus or growth. The course of the disease usually establishes the diagnosis.

**Course and Complications.** The skin often remains yellow after the bile pigment has disappeared from the urine. The jaundice generally

fades away in 2 or 3 weeks from the onset. Acute yellow atrophy of the liver is a rare complication

**Prognosis.** This is usually extremely good, acute yellow atrophy is a fatal complication

**Treatment** The patient should be kept in bed until the bile pigment has disappeared from the urine. A concentrated dose of mag sulph gr 60 to 120 in warm water fl oz 2 should be given every morning. If the bowels are not opened with the salts, an enema should be given. A salicylate mixture should be taken with the view to disinfecting the biliary tract, such as Sod salicyl gr 10, sod bicarb gr 20, syr aurant m 30 aquam ad fl oz 1. IT oz 1 t d s. The diet should be fat poor and liquid, or semi solid such as dextrose orangeade (dextrose 8 oz, water 2 pints and orange juice), barley water, meat extracts, potato, toast and rusks and  $\frac{1}{2}$  pint of milk daily. If the skin irritation is severe, the patient can be put in an alkaline bath (sod bicarb oz 8, water gallons 15) or the skin can be moistened with carbolic acid lotion (acid carbol m 3 aq ad fl oz 1)

### Epidemic Catarrhal Jaundice (Common Infective Hepatic Jaundice)

As the title suggests, two views are held regarding the pathology of non spirochaetal infective jaundice. 1 That it is due to duodenal catarrh. 2 That it is due to hepatitis which may proceed to hepatosis. Possibly both varieties exist. Epidemics are prone to occur in country districts and it has been suggested that even isolated cases of catarrhal jaundice are in reality sporadic manifestations of the infective disease, the contacts being immune.

Children and adults are affected, the incubation period being most frequently between 25 and 36 days. Infectivity persists for about 2 weeks close contact and possibly droplet infection being necessary for the spread of the disease. The clinical manifestations closely resemble those of catarrhal jaundice and all degrees of severity may be met with. In some cases petechial urticarial or morbilliform rashes appear. It is advisable to carry out tests to exclude the possibility of Weil's disease (see p 698)

### Acute Yellow Atrophy of the Liver (Acute Necrosis of the Liver)

**Definition** A disease characterised by progressive jaundice, fever severe nervous disturbances and shrinking of the liver

**Etiology** The cause is not known. The acute atrophy may occur in association with chemical poisons, such as chloroform, neoursphena mine alcohol phosphorus trinitro-toluene and tetrachlorethane. A few cases have resulted from the therapeutic administration of Atophan (cinchophenium B P). It is also met with in pregnancy and occasionally in such diseases as typhoid fever and influenza. **Predisposing causes** 1 Age. The majority of cases occur between the ages of 20 and 30, but it may develop at any age. 2 Sex. In adult life females predominate.

**Pathology** At autopsy the liver is small, greenish in colour and

the capsule is wrinkled. On section it is soft and yellow, due to excess of bile, and red areas caused by extravasated blood or hæmangiomata may be present. In cases showing a tendency to recovery, nodular hyperplasia of liver cells may be found. The spleen may be enlarged and soft, and meningeal hæmorrhages present.

**Clinical Findings.** The patient may be a pregnant woman, who is taken ill with the symptoms described on p. 87 as typical of catarrhal jaundice. In about 5 or 6 days, however, her condition becomes very much worse, the jaundice deepens, vomiting becomes intractable, and headache is severe.

**On Examination :** The patient is restless, muscular twitchings may be seen, and the tongue is brown and dry. The area of liver dulness is diminished, and it may be completely obliterated. The pulse is rapid, and the temperature is usually about 99° or 100° F. The pupils are often dilated, and the plantar response may be extensor. The bowels are constipated and the motions clay coloured. Hæmorrhages may occur from various sites, such as the stomach, intestines, kidneys, or under the skin. The urine is dark, containing bile pigments and usually some albumin and casts. Leucine and tyrosine crystals are also present. The blood shows a low alkali reserve, owing to the acidosis. It gives an immediate direct van den Bergh reaction.

**Differential Diagnosis.** At the onset the case resembles one of catarrhal jaundice, but in a few days it is obvious that the patient is gravely ill. Acute yellow atrophy may then be confused with spirochætal jaundice (see p. 698), but the liver is not diminished in the latter disease. In phosphorus poisoning also the liver is usually normal in size or somewhat enlarged.

**Course and Complications.** In severe cases the patient soon becomes delirious and comatose. The temperature may rise rapidly to 106° F. or higher just before death.

**Prognosis.** Death usually occurs in about 2 weeks in acute cases, in subacute cases it may be delayed for several weeks. Recovery may occur in the milder types of the disease.

**Treatment.** The patient must be kept quiet in bed, and the acidosis controlled by means of alkalis, dextrose and insulin. The dextrose can be given by mouth,  $\frac{1}{2}$  to 1 lb. daily in a quart of orangeade, or by rectal injections of 4 to 8 fl. oz. of normal saline containing 5% dextrose, every 4 to 6 hours, or by intravenous drip of 1 to 2 pints of normal saline, containing 5% dextrose, with 5 to 10 units of insulin twice a day. Sod. bicarb. gr. 60 to 120 should be given by mouth every 2½ hours. Plenty of fluids in addition should be taken such as water and barley water, and  $\frac{1}{2}$  to 1 pint of milk daily. The bowels should be kept open daily with salines, such as mag. sulph. gr. 60 to 120 mane, or with enemata. For insomnia and restlessness, bromides gr. 10 to 30 t.d.s. should be given. Morphine must not be administered owing to the risk of toxic effects.

### Tumours of the Liver

Simple and malignant tumours may arise in the liver. The simple tumours include an adenoma, angioma and teratoma. The adenoma

may be sufficiently large to be felt during life. An angioma usually gives rise to no symptoms or signs, and is discovered at autopsy. Teratomata are rare. The malignant tumours are either primary or secondary carcinoma or sarcoma. The majority of cases are secondary carcinoma.

### Primary Carcinoma

**Pathology** The following varieties are described. A large growth, distending the liver substance, smaller secondary deposits may occur in the liver. A diffuse growth which may be scirrhus. A multiple nodular growth. Cirrhosis carcinomatosa, in which the carcinoma probably develops in association with hepatic cirrhosis. Secondary deposits may be found in glands, the lungs, brain, bones, etc.

**Clinical Findings** The onset is insidious, the patient complaining of progressive weakness, with perhaps pain in the region of the liver. Frequently the tumour is not sufficiently large to be felt. There is usually no jaundice and often no ascites, but the patient rapidly goes down hill, and death occurs in a few months from the onset of the symptoms.

### Secondary Carcinoma

**Pathology** At autopsy the liver is enlarged and numerous deposits of whitish growth can be seen on the surface and on section. The projections may be umbilicated in the centre, owing to necrosis. Perihepatitis may be present in association with the nodules. The peritoneum may also be involved, with ascites. Dissemination to the liver is by the blood stream, but with a primary growth in the breast the spread occurs by lymphatic permeation. The primary growth is most frequently found in the stomach, then in the colon and rectum, the œsophagus, pancreas, gall bladder, uterus, breast, lungs, etc.

**Clinical Findings** The patient is most commonly a woman over the age of 40. There may be a definite history pointing to a carcinoma of the stomach, rectum or breast, or the first symptoms arousing attention may be hepatic in origin. In such a case it may be difficult to detect the site of the primary growth. The patient may complain of pains in the region of the liver, in the back, or down the arms. The skin may also itch very violently, although there is no icterus.

**On Examination** The abdomen is usually distended, whereas the body generally is wasted and the patient appears cachectic. The liver may be felt much enlarged, and the umbilicated nodules on its surface may be palpable. In other cases, where there is ascites, it may not be possible to feel the liver until the fluid has been removed. The spleen is usually not enlarged. Jaundice is not present in every case, but when it occurs it is usually obstructive in type and progressive. In addition to the ascites, in the later stages there is œdema of the feet, and dilated veins are seen in the lower part of the abdomen. A mass of growth may be felt near the umbilicus and enlarged glands may be present elsewhere, according to the general dissemination. There is usually a microcytic anaemia. The temperature is often irregularly raised.



**Differential Diagnosis.** This is usually quite clear when the primary growth can be detected. In other cases, such causes of hepatic enlargement as cirrhosis of the liver; gummatosis, amyloid degeneration, an abscess or hydatid cyst, a stone in the common bile duct, etc., must be considered. The Wassermann reaction should always be determined, and if found positive a course of iodides and mercury given (see p. 93).

**Course and Complications.** The patient becomes progressively more ill, and finally dies, in the majority of cases with cholæmic symptoms. There may be hæmorrhages in the skin, and cholangitis may occur as a complication.

**Prognosis.** Death generally takes place within a year from the diagnosis.

**Treatment.** This is only palliative. In some cases there is no pain, in others it is very severe. Relief can be obtained by aspirin gr. 10 t.d.s. by mouth, by Nепenthe m. 10 to 20 t.d.s. by mouth, or by subcutaneous injections of morphin. sulph. gr.  $\frac{1}{4}$  as required.

### Primary Sarcoma

This may form a single large tumour, or be found as multiple nodules, or as a diffuse growth. It is very rare.

### Secondary Sarcoma

The sarcoma is secondary to sarcoma of the adrenals, the mediastinum, the skin, a bone or the uveal tract in the eye. With melanotic sarcoma, melanin may be found in the urine, and the primary growth is in the skin or the eye.

### Hodgkin's Disease

This disease is more fully described on p. 507. When the liver is involved it is slightly enlarged, and ascites or jaundice may develop. The liver is firm and whitish in colour, the lymphogranulomatous tissue being spread throughout its substance.

### Amyloid Liver

**Etiology.** Amyloid liver occurs in association with chronic pulmonary tuberculosis, syphilis and long-standing cases of suppuration of the bones. It is comparatively rarely seen.

**Pathology.** The liver is enlarged, firm and smooth. It is tough to cut and pale on section. The amyloid degeneration is seen in the walls of the capillaries, especially in the intermediate zone of the lobules.

**Clinical Findings.** *On Examination:* The patient presents the signs of the disease which has led to the amyloid degeneration, such as long-standing pulmonary tuberculosis. The liver is enlarged and feels smooth and firm. There is no pain over the liver. Ascites is not common.

**Differential Diagnosis.** The firm character of the liver helps to differentiate it from the painless enlargement met with in fatty

## Actinomycosis of the Liver

**Etiology** The cause is the *Actinomyces bovis* (*Streptothrix actinomyces*) (see p 585)

**Pathology** The liver is usually infected secondarily to actinomycosis of the intestine. The lesion presents a honeycomb appearance and is bright yellow. There is often associated perihepatitis. The infection may spread directly to the pleura or lung, into the peritoneum, or work its way through the abdominal wall.

**Clinical Findings** The patient complains of ill health and malaise with fever. In addition there may be pain in the region of the liver.

**On Examination** The liver is palpable and tender, and the surface may be irregular. The blood shows a leucocytosis. If the actinomycotic lesion ulcerates through the abdominal wall, the streptothrix will usually be found in the pus.

**Prognosis** This is very grave.

**Treatment.** Sulphapyridine (M & B 693) should be administered 1 G t d s for 8 days followed by a second course 10 days later. If this is not successful potassium iodide should be given in doses up to gr 90 t d s. The local application of a radium pack together with diathermy is also worthy of trial.

## THE GALL-BLADDER

## Acute Cholecystitis

**Definition.** Acute inflammation of the gall bladder.

**Etiology** The inflammation results from bacterial infection. The organisms most often found are the *Bacterium commune* (*B. coli*), the *Bacterium typhosum* (*B. typhosus*) streptococci and staphylococci. Less frequently the *Diplococcus pneumoniae* (pneumococcus), the *Pseudomonas pyocyanea* (*B. pyocyaneus*) or anaerobes such as the *Clostridium welchii* (*B. welchii*) may be present. *Predisposing causes*

- 1 Age Usually over 40 years
- 2 Sex Females predominate
- 3 Constitution and habits Obesity, a sedentary life, and constipation.
- 4 Previous illness Especially typhoid fever and gall stones

**Pathology** The organisms may reach the gall bladder by various routes. The blood. Non-haemolytic streptococci may gain access to the gall bladder by the cystic artery coming from a distant focus in the tonsils or teeth. The intestines. The organisms are conveyed by the portal vein. The bile duct. The organisms such as the *B. typhosus* may ascend to the gall bladder against the bile stream. The lymphatics. The organisms may pass from the liver to the gall bladder.

Streptococci have been found in the submucous tissue of the gall bladder (mural cholecystitis) and in the cystic gland in a high percentage of cases, when they have not been present in the mucous membrane or in the contents of the gall bladder. These are presumably organisms which have been carried by the blood from a distant focus. The following varieties of acute cholecystitis are described. Catarrhal, suppurative, phlegmonous, gangrenous and membranous.

## Acute Catarrhal Cholecystitis

**Pathology.** The wall of the gall-bladder is inflamed, and adhesions may extend externally from its serous coat. The mucous membrane is swollen. The contents are clear, turbid or bile- or blood-stained fluid, and gall-stones may be present. The orifice of the cystic duct may be blocked by the swelling of the mucous membrane.

**Clinical Findings.** The patient complains of severe pain in the region of the gall-bladder. This may be paroxysmal and colicky in nature or a more continuous ache. The pain may radiate all over the abdomen, and to the right scapular region. There may be intense nausea and vomiting.

**On Examination:** The right upper rectus muscle is on guard, and tenderness is elicited over the gall-bladder. If there is cystic obstruction it may be possible to feel the distended gall-bladder. The temperature may be normal or slightly raised. Usually there is no jaundice.

**Differential Diagnosis.** It is impossible to eliminate with certainty the presence of gall-stones, but in biliary colic due to calculi the pain is of a more excruciating nature. An appendix abscess may closely simulate acute cholecystitis, but the swelling in the former is usually lower in the abdomen. With suppurative cholecystitis the constitutional disturbance is generally greater, and the temperature higher. In pyelonephritis the typical urinary changes are found (see p. 472).

**Course and Complications.** An acute attack may rapidly subside, but recurrence is not uncommon. Complications include suppurative cholecystitis, and perforation of the gall-bladder. Sequelæ include the formation of external adhesions, which may produce pyloric obstruction, and the development of chronic cholecystitis and gall-stones.

**Prognosis.** This is always uncertain in any particular case.

**Treatment.** The patient should be kept in bed, and pain relieved by the application of hot flannels over the gall-bladder. It may be necessary to inject subcutaneously morphin. sulph. gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  if the pain is very severe. The bowels should be opened and the gall-bladder encouraged to empty itself by giving mag. sulph. gr. 60 to 120 in hot water, fl. oz. 2 every morning. A course of hexamine and alkalis should be given, as for chronic cholecystitis (see p. 97). The diet must be fluid during the acute stage, such as milk and soda, and meat extracts. *If the vomiting is severe, a mixture containing Bism. carb. gr. 15, sod. bicarb. gr. 10, acid. hydrocyan. dil. m. 3, sp. chlorof. m. 5 and aq. menth. pip. dest. ad fl. oz. 1 should be given 3 or 4 times a day.*

## Suppurative Cholecystitis

**Pathology.** The wall of the gall-bladder may show ulceration. The gall-bladder may be distended, or shrunken from previous cholecystitis. It contains purulent fluid, and gall-stones are often present. A condition of chronic empyema of the gall-bladder may occur (see p. 98).

**Clinical Findings.** The patient is acutely ill, the pain is very severe and often paroxysmal. There may be sweating and rigors. The blood usually shows a leucocytosis of over 15,000 per c.mm. The temperature

and pulse are raised, and there is extreme tenderness on palpation over the gall bladder. There may be slight jaundice.

**Differential Diagnosis** The condition may resemble an abdominal emergency such as a perforated duodenal ulcer or appendix, or sub-phrenic abscess. The diagnosis depends upon the signs being localised to the gall bladder region.

**Course and Complications** The course is usually rapidly progressive, unless relieved by operation. Complications include perforation, with local or generalised peritonitis. The gall bladder may be adherent to surrounding structures and thus perforate into the stomach, duodenum or intestine, or into the pleura or kidney pelvis. A liver abscess or cholangitis may occur as complications.

**Prognosis** This is grave, unless rapidly improved by operation.

**Treatment** An operation should be performed and the gall bladder removed.

### Phlegmonous Cholecystitis

This is a severe form of suppurative cholecystitis, which requires immediate operation.

### Gangrenous Cholecystitis

Gangrene of the gall bladder is a sequel of suppurative or phlegmonous cholecystitis. It resembles clinically acute peritonitis. Treatment is by operation.

### Membranous Cholecystitis

Casts may form in the gall bladder and be passed in the motions. The symptoms resemble those of biliary colic due to calculi. Treatment is by cholecystectomy.

### Subacute Cholecystitis

(*Lipoid Cholecystitis Cholesterosis 'Strawberry Gall bladder'*)

**Pathology** The gall bladder is usually normal externally, but the cystic gland is enlarged. Yellowish white spots are dotted all over the mucous membrane. These are due to deposition of lipoid (cholesterol ester) in the cells of the mucosa. Mulberry cholesterol stones may be present. Streptococci are usually found in the submucous tissue and in the cystic gland.

**Clinical Findings.** The symptoms closely resemble those of catarrhal cholecystitis, but are of a milder degree than those described under the acute catarrhal infection.

**Treatment** The gall bladder should be removed.

### Chronic Cholecystitis

**Etiology** Chronic cholecystitis is due to infection with similar types of organisms as described for acute cholecystitis. It may develop insidiously or follow an acute attack.

**Pathology** The wall of the gall bladder is often thickened, with external adhesions. In some cases, if a calculus is occluding the cystic

duct, the gall-bladder is distended. Calculi are often present and the cystic gland is enlarged.

**Clinical Findings.** The patient complains of chronic indigestion, the chief features of which are flatulence and epigastric distention after meals and periodical attacks of nausea or vomiting. Pain may be felt also in the region of the gall-bladder or in the epigastrium, and may radiate around the chest and pass to the right scapular region. There may be slight icterus with fever from time to time.

**On Examination :** The right upper rectus is usually slightly rigid as compared with the left. On palpation in the region of the gall-bladder the patient may experience pain on taking a deep breath, and the breathing may be suddenly checked (Murphy's sign). This may be best elicited if the patient is examined standing and bending slightly forward. A cholecystogram may show that the gall-bladder does not fill or contract normally, or the gall-bladder when thus visualised by the X-rays may be found to be tender on palpation. Bile removed by Lyon's method with duodenal intubation may be unduly pale, and cells or organisms may be present. There may be achlorhydria.

**Differential Diagnosis.** Chronic cholecystitis may be confused with a gastric or duodenal ulcer, with gall-stones, with chronic appendicitis, or with angina pectoris. The pain referred from arthritis of the spine is also misleading. The opaque meal affords valuable indication as to the presence of gastric or duodenal ulcers. Gall-stones may be shown by direct X-ray or by the cholecystogram, but they are usually associated with chronic cholecystitis.

Chronic appendicitis also may be present with chronic cholecystitis, but palpation over the appendix when visualised by X-rays usually causes pain in chronic appendicitis. An X-ray examination of the mid-thoracic spine will exclude arthritis.

**Course and Complications.** Chronic cholecystitis, unless adequately treated, is usually a progressive lesion. An attack of acute cholecystitis may occur at any time. Complications include the formation of gall-stones with biliary colic, pancreatitis, arthritis, phlebitis and myocardial degeneration.

**Prognosis.** Chronic cholecystitis is a cause of persistent ill-health, but is not usually a fatal disease.

**Treatment.** Any focus of sepsis in the teeth and naso-pharynx should be removed. If gall-stones are present, or if there is evidence of chronic appendicitis, the gall-bladder and appendix should be removed. In other cases an attempt should be made to disinfect the contents of the gall-bladder by means of hexamine, which, in the presence of bile, is potent in an alkaline medium. Two mixtures are ordered, Hexamine gr. 100, aq. ad fl. oz. 1. and Pot. cit. and sod. cit.  $\overline{\text{aa}}$  gr. 100, aq. ad fl. oz. 1. The patient begins the course with m. 60 of the hexamine mixture and 1 fl. oz. of the alkaline mixture after breakfast, tea, and after a glass of milk or water last thing at night. The hexamine mixture is increased by m. 60 daily until the patient is taking fl. oz. 1 (gr. 100) t.i.d. The urine should be tested 3 times a day, as unless an alkaline reaction is maintained there is a risk of producing vesical irritability

and hæmaturia. This dose is continued for 5 or 6 weeks when the symptoms should have disappeared. On waking, mag sulph gr 30 to 120 should be taken in 2 fl oz of hot water, an hour before breakfast, and the patient should then lie on the right side. This should cause the gall bladder to contract. The amount of mag sulph taken is regulated by its effect on the bowels, as it is not desired that there should be diarrhoea. There is no necessity to give a fat-poor diet if there is no evidence of cholelithiasis.

### Chronic Empyema of the Gall-bladder

This may be a sequel of acute cholecystitis. The patient complains of pain and tenderness in the region of the gall bladder, which may be palpable. There is usually no fever.

### Torsion of the Gall-bladder

This is a rare condition resembling clinically an acute abdominal emergency. It may result in gangrene of the gall bladder. Treatment consists in cholecystectomy.

### Parasitic Infections of the Gall-bladder

These are rare. At times the *Ascaris lumbricoides*, *Lambia intestinalis*, *Echinococcus granulosus* (*T. echinococcus*) or the *Distoma hepaticum* may be present in the gall bladder.

### Tumours of the Gall-bladder

*Simple Tumours* These are rare and cannot usually be diagnosed. They include papilloma, adenoma and fibroma.

*Malignant Tumours* These include primary and secondary carcinoma and sarcoma. The majority of cases are primary carcinoma.

### Primary Carcinoma of the Gall-bladder

*Etiology* The cause is not known, but the growth is often associated with gall stones or chronic cholecystitis. Other predisposing causes include—1 Age Usually over 45. 2 Sex Females predominate in the proportion of about 4 to 1.

*Pathology* The growth may be papillomatous and fungating or diffuse and infiltrating. It is most often situated at the fundus. It may spread directly to the liver, into the bile ducts, or form a sinus through the abdominal wall or a fistula into the colon. It may perforate intraperitoneally. Thrombosis of the portal vein may be found, and secondary deposits in the liver, or in lymph glands in the portal fissure, above the clavicles, or in the anterior mediastinum. Microscopically the growth may be columnar or spheroidal celled and colloid changes may be present.

*Clinical Findings.* The patient is often a woman over the age of 45, who gives a history suggestive of chronic cholecystitis or gall stones. More recently she has noticed increasing discomfort or pain in the region of the gall bladder, with loss of strength.

**On Examination :** In the early stages nothing may be found, but later the gall-bladder is palpable, tender, and the surface is often irregular. The liver may also be enlarged. If there is pressure on the bile ducts, either from the growth or from the enlarged portal glands, there will be obstructive jaundice. There may also be ascites and swelling of the legs. Enlarged glands may be felt above the clavicles. In the later stages cachexia is more marked, and hæmorrhages may be seen under the skin. A cholecystogram may reveal a filling defect in the gall-bladder.

**Differential Diagnosis.** In the early stages the symptoms usually suggest cholecystitis or cholelithiasis. Later it is often difficult to differentiate carcinoma of the gall-bladder from a growth in the liver, pancreas or bile ducts. An opaque meal should serve to exclude carcinoma of the stomach.

**Course and Complications.** The course is usually rapidly progressive. Such complications as perforation, fistula formation or suppurative cholangitis may occur.

**Prognosis.** The disease is usually fatal within 8 months from the onset of symptoms.

**Treatment.** This is only symptomatic and palliative, as operative removal of the growth is usually impossible.

### Secondary Carcinoma of the Gall-bladder

The gall-bladder may be affected secondarily by direct spread from a growth of the stomach or colon, or by metastases from growths elsewhere. These deposits are usually subperitoneal.

### Gall-stones

#### (Cholelithiasis)

**Definition.** Calculi formed in the biliary passages or gall-bladder.

**Etiology.** The main factors in their formation are: Infection, biliary stasis, and hypercholesterolemia. *Predisposing causes :* 1. Age: Usually over 40. 2. Sex: Females predominate. 3. Habits: A sedentary life and over-eating. 4. Associated conditions: Pregnancy, chronic diseases of the heart or lungs, constipation. 5. Climate: Especially the temperate zones. 6. Heredity: There is a familial tendency.

**Pathology.** Frequently cholecystitis precedes the formation of calculi. A central nucleus consisting of organisms (not infrequently anaerobes), mucus or fibrin may be the starting-point. The bile in the gall-bladder, especially if stagnant, may be unduly rich in pigment, cholesterol or lime salts. The gall-bladder wall is frequently infected, even when its contents are sterile. Thus in a series of cases of gall-stones, the gall-bladder was infected in 70%, the fluid contents in 40% and the stones in 30% of the cases. Aseptic calculi are thought to occur apart from infection, especially when the blood cholesterol is high, as in pregnancy and some cases of obesity and arteriosclerosis. A solitary calculus may be found, or as many as 14,000 stones may be present in the gall-bladder. The stones may be situated in the cystic duct, or less commonly in the common bile duct and the extra- or intra-hepatic ducts.

*Varieties* 1 The pure cholesterol stone This is usually solitary, oval or circular, pale and very light It may be formed apart from sepsis

2 The laminated cholesterol stone containing layers of cholesterol and calcium bilirubin

3. Mixed gall stones containing cholesterol (80% to 98%) and calcium bilirubin These are soft before they are dried

4 Pure calcium bilirubin (mulberry stones) Small hard irregular stones

5 Calcium carbonate These stones are rare

*Clinical Findings* In some cases stones in the gall bladder give rise to no symptoms, and are only demonstrated post mortem In other cases the symptoms are those described above as being typical of chronic cholecystitis If the stone becomes impacted in the cystic duct, the gall bladder enlarges and is painful, but there is no jaundice When the calculus enters the common bile duct biliary colic usually ensues According to Courvoisier's law, the gall bladder is usually not distended in cases of jaundice due to a calculus in the common bile duct, owing to the presence of old cholecystitis, whereas in obstruction of the common bile duct due to a growth the gall bladder is dilated In some cases there is persistent jaundice without pain If the stone is lodged in the ampulla of Vater, forming a ball valve obstruction, there are usually periodical attacks of fever and jaundice, known as *the intermittent hepatic fever of Charcot*

*Biliary Colic* This may occur when the stone enters one of the biliary passages The onset of the symptoms is often sudden, occurring frequently during the night The patient complains of excruciating pain which radiates all over the abdomen to the right scapular region and tip of the right shoulder It is paroxysmal, the patient rolls about or doubles up in agony There is sweating and often vomiting Attacks of biliary colic not due to a gall stone are described These may occur after cholecystectomy They are thought to be due to achalasia of the sphincter of Oddi

*On Examination* It is not usually possible to feel the gall bladder, but the right upper rectus is somewhat rigid The temperature may be normal or slightly raised The pulse is feeble, but is not increased in rate Jaundice may be noted a day or so after the attack, and stones may be found in the faeces Whether or not gall stones can be seen by X rays depends upon their calcium content A cholecystogram may reveal their presence as a lighter shadow in the opaque gall bladder when they are not demonstrable in a direct radiogram

*Differential Diagnosis* Biliary colic must be differentiated from renal, pancreatic or intestinal colic, a tabetic crisis associated with colic and jaundice, the pain of coronary thrombosis, of acute pancreatitis or perforation of a gastric or duodenal ulcer The excruciating paroxysmal pain with its characteristic distribution usually serves to differentiate

*Course and Complications* The attack may last from a few minutes to a few hours Recurrences are liable to occur Complications include perforation of the gall bladder, ulceration into the small intestine with subsequent intestinal obstruction, formation of various fistulae such as



a gastric, duodenal, intestinal, colic, or bronchial fistula, or a cutaneous sinus. Suppurative cholangitis or liver abscess may also ensue. Intestinal volvulus, acute or chronic pancreatitis, or carcinoma of the gall-bladder or bile ducts may follow.

**Prognosis.** Death during an attack is rare, but succeeding attacks are liable to occur, and gall-stones may form in the biliary passages after cholecystectomy.

**Treatment.** *Prophylactic.* Chronic cholecystitis should be treated as described above. If there is a tendency to hypercholesterolaemia, the diet should be fat-poor, and eggs should not be eaten.

*Curative.* During an attack of biliary colic the acute pain must be controlled either by the subcutaneous injection of morphine sulphate gr.  $\frac{1}{4}$  and atropine sulphate gr.  $\frac{1}{100}$ , by the intravenous injection of atropine sulphate gr.  $\frac{1}{100}$ , or by the inhalation of chloroform. Milder attacks may be mitigated by placing the patient in a hot bath and giving by mouth tnc. belladon. m. 30, repeated in an hour if necessary. Subsequently the gall-bladder may be removed, or the patient may receive a course of treatment as for chronic cholecystitis. There is no known method of dissolving gall-stones.

## THE BILE DUCTS

### Congenital Obliteration

This is often associated with cirrhosis of the liver, and jaundice is present either at birth or a few weeks later. The liver and spleen are usually palpable; bile pigment is present in the urine, but not in the stools. Haemorrhages may occur in the skin, mucous membranes or internal organs. The condition is not syphilitic. Death usually occurs within a few weeks.

Congenital syphilitic stricture of the bile ducts is rare.

### Suppurative Cholangitis

**Etiology.** Suppurative cholangitis may be associated with gall-stones, cholecystitis or obstruction of the extra-hepatic ducts by a tumour. It may occur as a complication of such diseases as pneumonia or enteric fever, or be secondary to an abscess in the liver.

**Pathology.** Suppuration may be found in the extra-hepatic bile ducts and the gall-bladder. The liver is enlarged and numerous small abscesses may be present.

**Clinical Findings.** The chief features are fever with jaundice and rigors. The patient is very gravely ill and the liver is usually enlarged and tender.

**Differential Diagnosis.** It is often impossible to diagnose suppurative cholangitis from suppurative pyelophlebitis. It is distinguished from intermittent hepatic fever (see p. 100) by the intervals of comparative health in the latter disease.

**Course and Complications.** The disease is usually progressive. Complications include pancreatitis and pyaemia.

**Prognosis.** This is always very grave, and the disease is fatal when abscesses form in the liver.

**Treatment.** An operation may enable the extra hepatic ducts to be drained

### Chronic Catarrhal Cholangitis

This may be associated with cholecystitis and gall stones or occur as a sequela of chronic catarrhal jaundice. The extra hepatic ducts in these cases are usually affected. In cirrhosis of the liver the intra-hepatic ducts may be involved.

### Calculi in the Bile Ducts

A description of the effects produced by calculi in the bile ducts is given on p. 100.

### Tumours of the Bile Ducts

**Simple Tumours.** These include a papilloma, adenoma and fibroma. They are all rare.

**Malignant Tumours.** **Pathology.** Primary carcinoma of the extra hepatic ducts appears in the following sites in this order of frequency: At the union of the common hepatic and cystic duct, at the lower end of the common bile duct, in the common hepatic duct, in the cystic duct. The gall bladder is dilated with growths in the cystic duct and in the common bile duct but there is no jaundice in the former case. The growth may spread directly into the pancreas. In some cases the growth originates in the gall bladder and spreads down the lumen of the bile duct.

**Clinical Findings.** The patient is usually an adult male. The onset of the disease is often insidious with jaundice, which becomes more intense, progressive weakness and irregular fever. There is usually no pain.

**On Examination.** The growth cannot be felt, but the gall bladder may be palpable. Bile is usually present in the urine, and the faeces are pale.

**Differential Diagnosis.** It is often difficult to distinguish a tumour of the bile duct from carcinoma of the head of the pancreas, and in other cases from a stone blocking the cystic duct.

**Course and Complications.** The course is steadily progressive, and cholaemia occurs as a terminal phase.

**Prognosis.** The patient usually dies in a few months.

**Treatment.** An exploratory laparotomy is usually performed to establish the diagnosis, and in some cases it is possible to relieve the jaundice by means of cholecystenterostomy.

## THE PERITONEUM

### Acute Peritonitis

**Definition.** Acute inflammation of the peritoneum.

**Etiology.** Acute peritonitis results from bacterial infection. The causative organisms include streptococci, the *Bacterium commune* (*B. coli*), staphylococci, the *Mycobacterium tuberculosis* (*B. tuberculosis*),

the *Diplococcus pneumoniae* (pneumococcus), the *Neisseria gonorrhoea* (gonococcus), the *Bacterium friedländeri* (pneumobacillus), the *Pseudomonas pyocyanea* (*B. pyocyaneus*), the *Bacterium typhosum* (*B. typhosus*) and anaerobic organisms.

**Pathology.** The organisms usually reach the peritoneum from the alimentary tract, either through a spot of lowered resistance in the wall, or through an actual perforation. They may also gain access from the gall-bladder, a liver abscess, the uterus, or through the Fallopian tubes. In some cases they may be blood-borne, or enter through a wound of the abdomen. Terminal peritonitis may occur in chronic nephritis. The peritonitis may be localised, as around the appendix and in the pelvis, or diffuse. The inflammation may be fibrinous or exudative, and adhesions usually form after removal of the exudate. The exudate may be serous, purulent or hæmorrhagic, and contain gas. There is usually intestinal paralysis. Certain varieties are described, according to the primary infecting organism: Streptococcal, staphylococcal and *B. coli* infections.

**Clinical Findings.** The patient is suddenly seized with agonising abdominal pain which increases in severity. He vomits, and the bowels may be freely opened at first, but soon are constipated.

**On Examination:** The patient is usually found lying on his back with his knees drawn up; the breathing is shallow. Abdominal movement is absent over the affected part of the abdomen. The abdominal wall is rigid, either locally or generally, and tender to light touch. The abdomen becomes distended. The liver dulness may be diminished if gas has escaped from the alimentary tract. No intestinal movements can be detected with the stethoscope. The facial expression is drawn and anxious (*facies Hippocratica*). The temperature may be a little raised or sub-normal, but the pulse is frequent and of small volume. The tongue becomes furred and dry. A leucocytosis occurs in the course of a few hours.

**Differential Diagnosis.** Acute peritonitis may be mistaken for intestinal colic, obstruction or hæmorrhage, a ruptured tubal pregnancy, acute appendicitis without perforation, acute hæmorrhagic pancreatitis, mesenteric thrombosis, a tabetic crisis, or hysteria. The important features in acute peritonitis are the absence of abdominal movement, and the rigidity. These may, however, not be very evident in a case in which the peritonitis has been present for many hours. The temperature may also be subnormal. The pulse is a good guide, as it becomes progressively more frequent as the condition deteriorates, and the tongue becomes more dry.

**Course and Complications.** If untreated, death occurs in the course of a day or so with generalised peritonitis, but there may be a short period of temporary improvement which is deceptive. Localised peritonitis may become completely shut off by adhesions and heal spontaneously.

**Prognosis.** This is always very grave in general peritonitis, and death occurs in a few days if no operation is performed.

**Treatment.** Immediately the diagnosis is made and an operation

decided upon, a subcutaneous injection of morphin sulph gr  $\frac{1}{4}$  to  $\frac{1}{2}$  may be given to relieve pain. The curative treatment is surgical.

### Pneumococcal Peritonitis

**Pathology** The infection usually spreads from the vagina to the uterus, tubes and peritoneum. In some instances the pneumococci are blood borne from a focus elsewhere such as otitis media, and it is doubtful whether pneumococci do at times enter the peritoneum from the intestine. The peritonitis is usually diffuse, but may be localised. The pus is thick, yellowish green, flaky and usually without smell.

**Clinical Findings** The patient is generally a girl under the age of 10 years. She is suddenly taken ill with severe abdominal pain, vomiting and diarrhoea.

**On Examination** The tongue is furred and dry. The temperature is raised to 103° F or higher, the pulse is frequent and the respirations may be rapid. The abdomen is distended especially around the umbilicus and the movement is slight. The abdomen feels doughy, is moderately rigid and universally tender.

**Differential Diagnosis** Pneumonia is excluded by the absence of pulmonary signs. With acute appendicitis the pain usually begins near the umbilicus, and when peritonitis is present the signs are more localised to the right iliac fossa.

**Course and Complications** The vomiting usually persists, but the bowels tend to become costive.

**Prognosis** In some cases recovery occurs apart from operation, but the outlook is very grave in children.

**Treatment** Operation is usually advisable in order to establish the diagnosis. Sulphapyridine (M & B 693) should be administered in doses according to the age of the patient. For a child of 10 years 1 G should be given every 4 hours for 24 hours, followed by 0.5 G 4 hourly for 2 to 3 days.

### Acute Tuberculous Peritonitis

**Etiology** Acute tuberculous peritonitis is generally secondary to tuberculosis of the abdominal glands, the intestines, or genital organs.

**Clinical Findings** The onset may closely simulate that of typhoid fever, the patient being taken ill with headache, malaise and abdominal discomfort. The bowels are loose or constipated.

**On Examination** The temperature is raised, but often the pulse is not proportionally frequent. The temperature assumes the continuous type, with morning falls and evening rises of a degree or so. Gradually the abdomen becomes distended, first with flatus and later fluid may be detected. Definite swellings due to matted omentum or glands may be felt.

**Course and Complications** In a favourable case the temperature gradually falls to normal in the course of a few weeks, complications such as a pleural effusion, intestinal obstruction, tuberculous enteritis, etc., may occur.

**Treatment.** The patient must be kept in bed during the febrile stage. Artificial or natural sunlight treatment should be given, starting with an exposure of 10 minutes daily to the feet, and gradually exposing the whole of the legs, for periods up to 1 or 1½ hours daily. Ung. hydrarg. co. gr. 60 spread on lint may be applied to the abdomen for 4 successive days each week. If the fluid still increases in amount, a laparotomy with evacuation of the fluid often accelerates recovery, or the fluid may be removed by paracentesis and some air inserted into the peritoneal cavity with a pneumothorax apparatus.

### Gonococcal Peritonitis

This is usually secondary to infection of the Fallopian tubes; less often it occurs as a complication of gonococcal epididymitis. The peritonitis is generally localised to the pelvis, and it often resolves without operation.

### Subdiaphragmatic Abscess

#### (*Subphrenic Abscess*)

**Definition.** A localised variety of acute peritonitis, with suppuration between the liver and diaphragm.

**Anatomy and Etiology.** Six anatomical varieties are recognised, according to the relation of the abscess to the hepatic ligaments; four of these varieties are intraperitoneal, and two are extraperitoneal (see Fig. 4). The reader should consult Fifeild and McNeill Love (*Brit. Journ. Surg.*, 1926, 13, 683), on whose article this section is largely based.

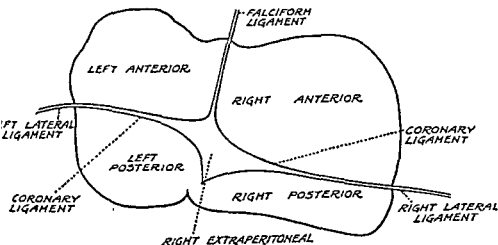


FIG. 4. DIAGRAM: SUPERO-POSTERIOR VIEW OF LIGAMENTS OF LIVER, SHOWING SITES OF SUBPHRENIC ABSCESS.

1. *Right Anterior Intraperitoneal.* The pus collects between the right side of the diaphragm and the superior, anterior and right lateral surfaces of the right lobe of the liver. In front there are the diaphragm and the abdominal wall. Behind the space is bounded by the anterior layer of the coronary ligament and the right lateral ligament. Above is the diaphragm. Below adhesions form between the transverse colon

and great omentum, and the anterior abdominal wall. On the right are the abdominal wall and the diaphragm, and on the left the falciform ligament. The infection is derived from an appendix abscess, a perforated gastric or duodenal ulcer, or from suppuration in the liver or bile ducts.

**2 Right Posterior Intrapertitoneal** (Cantlie's subhepatic pouch, Rutherford Morrison's kidney pouch). The pus collects in a pyramidal space, which lies transversely beneath the right lobe of the liver, with its apex medially disposed, close to the foramen of Winslow. In front there are the liver and gall bladder, behind the diaphragm and upper part of the right kidney, above are the liver, the posterior layer of the coronary ligament and the right lateral ligament, below lie the transverse colon and the hepatic flexure. On the right there are the right lobe of the liver and the diaphragm, and on the left the foramen of Winslow and the duodenum. The infection originates in the appendix, and rarely from a perforated gastric or duodenal ulcer, from a liver abscess or from suppuration in the thorax.

**3. Left Anterior Intrapertitoneal.** The abscess space is bounded by the following structures. In front are the diaphragm and anterior abdominal wall, behind lie the left lateral ligament, the left lobe of the liver, the gastro-hepatic omentum and the anterior surface of the stomach. Above is the diaphragm. Below adhesions form between the stomach or great omentum and the anterior abdominal wall or diaphragm. On the right is the falciform ligament, and on the left the spleen, gastro-splenic omentum and diaphragm. The pus is derived from a perforated gastric or duodenal ulcer, or from a splenic, hepatic or pelvic abscess.

**4 Left Posterior Intrapertitoneal.** The abscess is in the lesser sac of the peritoneum, the foramen of Winslow being closed by adhesions. In front the space is bounded by the caudate lobe of the liver, the gastro-hepatic omentum, the stomach and the two anterior layers of the great omentum. Behind there are the diaphragm, the pancreas, the transverse meso-colon and the two posterior layers of the great omentum. Above is the coronary ligament. Below is the fusion between the layers of the great omentum. On the right are the union of the layers of the great omentum and the first part of the duodenum. On the left there lie the lienorenal ligament, the spleen, the gastro-splenic omentum, and the junction of the layers of the great omentum. The abscess is derived from a perforated posterior gastric ulcer, a splenic, hepatic or pancreatic suppuration, or from spread of infection in general peritonitis.

**5 Right Extrapertitoneal.** Suppuration occurs between the diaphragm and the bare area of the liver. In front there are the right lobe of the liver and the right suprarenal. Behind is the diaphragm. Above is the anterior layer of the coronary ligament, below is the posterior layer of the coronary ligament. To the right lies the right lateral ligament, and to the left the inferior vena cava and right crus of the diaphragm. The abscess may extend forwards to the epigastrium between the layers of the falciform ligament. The infection is due to suppuration in the liver or bile ducts, or a spread from the abdominal wall by the lymphatics in the falciform ligament, or from the right

retroperitoneal tissue in a perforated posterior duodenal ulcer, a retrocaecal appendix, ulceration of the ascending colon or hepatic flexure, or a perinephric or pancreatic abscess. It may be secondary to suppuration above the diaphragm.

6. *Left Extraperitoneal.* Suppuration occurs in a potential space formed by stripping the peritoneum off the left side of the diaphragm. *In front are the bare area of the stomach, pancreas and spleen. Behind* are the upper portion of the left kidney and the suprarenal. Above is the diaphragm. Below, the abscess can extend downwards for a variable distance retroperitoneally. To the right are the aorta and vertebræ, to the left is the diaphragm. The suppuration may be secondary to a left perinephric abscess, a perforated posterior gastric ulcer, ulceration of the colon, diverticulitis, acute osteomyelitis of the lumbar vertebræ, perforation of the œsophagus, or to suppuration above the diaphragm.

**Clinical Findings.** Subdiaphragmatic abscess is usually secondary to suppurative appendicitis or to perforation of the stomach or duodenum. There may be a history of an operation from 4 to 7 months previously. The temperature and pulse rate then rise, and the patient becomes more ill. Rigors, sweating and pain in the region of the diaphragm or shoulder may develop.

**On Examination:** The patient looks ill. If the abscess is anterior a bulging or swelling may be seen under the costal margin, it is dull on percussion, and the chest moves little on the affected side. If the stomach or duodenum have perforated, gas may also be present in the abscess, and a resonant note may be obtained over a small area, which keeps uppermost as the patient is turned from his back to his side. Abnormal signs are found at the base of the corresponding lung, for the diaphragm is raised and may be as high as the second or third rib. Typically four zones can be detected at the back of the chest, at the apex the normal lung, below this is a zone of collapsed lung with some dulness and weak air entry, still lower there is a tympanitic note due to gas in the abscess, and at the base there is liver dulness. When the abscess is a right posterior one, a swelling may be found in the region of the right loin. With a left posterior abscess there may be no swelling seen, or one may be detected in the abdomen above or below the stomach. With an extraperitoneal abscess on the bare area of the liver, the liver is usually displaced downwards, the right diaphragm is raised, and there is dulness and deficient air entry over the lower lobe of the right lung. The blood: There is usually a leucocytosis of 15,000 to 20,000 per c.mm. An X-ray examination may show the position of the diaphragm and of the liver. Exploratory puncture through the 9th, 10th or 11th intercostal space in the line of the vertebral border of the scapula may reveal the abscess, the diaphragm being pierced by the needle before the pus is struck, and so oscillating with respiration.

**Differential Diagnosis.** A subphrenic abscess is often difficult to diagnose; it may be mistaken for septicæmia or pyæmia, empyema, pneumothorax, perinephric abscess, liver abscess, pylephlebitis, or a

pancreatic cyst The history, X ray examination and results of exploratory puncture usually serve to establish the nature of the condition

**Course and Complications** If left untreated, the abscess may cause a secondary pleural effusion or it may rupture into the pleura or lung, with resultant empyema or lung abscess Rupture may also occur into the pericardium, œsophagus, the general peritoneal cavity, or through the skin Septicæmia or pyæmia may occur as further complications

**Prognosis** Subphrenic abscess is a serious condition, with a mortality of about 70% if left undrained, which is lowered by about a half if adequate treatment is given

**Treatment.** The abscess should be drained surgically, but the exploratory puncture should be performed in the theatre by a surgeon prepared to complete the operation

### Chronic Peritonitis

**Definition** Chronic inflammation of the peritoneum

**Etiology** Chronic peritonitis may follow acute peritonitis or occur more insidiously The following varieties may be recognised —

#### Localised, Plastic or Proliferative Peritonitis

**Etiology** Localised peritonitis may occur after abdominal operations or secondary to inflammation of the appendix, gall bladder, Fallopian tubes, abdominal glands, diverticula, etc In other cases it is a form of chronic tuberculous peritonitis Proliferative peritonitis may be localised round an organ such as the spleen or liver The latter may be covered with a thick white coat of fibrous tissue (‘sugar iced liver’ see p 74)

#### Diffuse Plastic or Proliferative Peritonitis

**Etiology** Diffuse peritonitis may occur as a form of chronic tuberculous peritonitis with carcinoma of the peritoneum or as an extension of a chronic plastic peritonitis of septic origin In Pick’s disease there is a diffuse proliferative peritonitis often associated with similar proliferative changes in the mediastinum pericardium and pleura (see *Chronic Indurative Mediastino pericarditis* p 210) and with chronic nephritis and arteriosclerosis In Concato’s disease the peritoneum is much thickened the omentum is often rolled up and there is chronic perihepatitis or perisplenitis A serous effusion may be present, and when the pericardium and pleuræ are involved the condition is known as polyserositis or polyorrhomentitis A barium meal may show a peculiar condition of thickening of the stomach and the cæcum and colon may also be involved

#### Chronic Tuberculous Peritonitis

**Etiology** The *Mycobacterium tuberculosis* (B tuberculosis) usually gains access to the peritoneum from the intestine the mesenteric and retroperitoneal glands It may, however, be a blood borne infection



from a focus in any part of the body. Cirrhosis of the liver is a predisposing cause in adults.

**Pathology.** Certain types are described, which tend to overlap each other. 1. *Tabes mesenterica*: The mesenteric and retroperitoneal glands are affected. 2. *The plastic or fibroid variety*: Adhesions form, the intestines are matted together, the omentum may be rolled up in a mass, and there is no effusion. 3. *The ascitic form*: The fluid may be free or loculated by adhesions. 4. *The caseous variety*: Softening occurs in the tuberculous foci, there may be localised suppuration and the formation of *fæcal fistulæ*.

**Clinical Findings.** The patient is usually between the ages of 3 and 25 years. The onset is insidious; thus the child is gradually taken ill with lassitude, loss of appetite, pallor, abdominal discomfort or colic. The bowels are usually constipated.

**On Examination:** The child looks somewhat wasted, but the abdomen is often distended. The skin, especially over the abdomen, may be pigmented. In the plastic variety the abdomen has a peculiar doughy feeling. Small masses may be felt, due to glands or matted omentum. Loculated fluid gives rise to a dull swelling which may simulate an ovarian cyst. When there is free fluid the signs are those of ascites (see p. 111). The temperature is usually slightly raised and irregular, but it may be normal. The bowels are costive in the plastic variety, but with *tabes mesenterica*, obstruction of the lacteals may result in diarrhoea with fatty stools.

**Differential Diagnosis.** Chronic tuberculous peritonitis may be mistaken for other causes of wasting, coeliac disease, chronic appendicitis, regional ileitis, salpingitis, an ovarian cyst, abdominal carcinoma and cirrhosis of the liver or other causes of ascites. Diagnosis is established in the ascitic variety by removing a specimen of the fluid and injecting it into a guinea-pig, in which tuberculous lesions develop in 2 to 3 months. An X-ray examination which reveals calcified abdominal glands is in favour of the presence of unhealed tuberculous glands in the abdomen.

**Course and Complications.** The course is prolonged in the majority of cases. Complications include intestinal obstruction or perforation, *fæcal fistula* formation, localised suppuration, and general dissemination by the blood stream.

**Prognosis.** There is usually a tendency to recovery; the most unfavourable developments are the general dissemination of tuberculosis, intestinal obstruction, the formation of *fæcal fistulæ* or of local abscesses.

**Treatment.** This is usually medical. The patient should be kept in bed during the febrile period, preferably in the open air. Exposure to sunlight, real or artificial, should be carried out as for acute tuberculous peritonitis (see p. 103). The diet should be of a good nourishing variety, and cod-liver oil in doses of m. 30 to 60 daily should be taken periodically when the weather is not hot. Operation is required for intestinal obstruction. Ascites should be dealt with as in acute tuberculous peritonitis (see p. 103).

### Cysts of the Peritoneum

The cysts may be mesenteric (see p 112), hydatid, dermoid or teratomatous. They cause vague symptoms, such as abdominal discomfort and swelling, and the treatment is surgical.

### New Growths of the Peritoneum

These are simple and malignant. Simple growths include a fibroma, lipoma, angioma, lymphangioma, and myoma. Malignant growths may be primary, such as an endothelioma or sarcoma. Usually they are secondary to carcinoma of the ovary, stomach, intestine or breast or to a pleural endothelioma. The peritoneum may be studded with minute nodules and an effusion, serous, hæmorrhagic or chylous may form.

### Ascites

(including *Hydroperitoneum*, *Seroperitoneum*, *Hæmoperitoneum* and *Chyloperitoneum*)

**Definition.** Non purulent fluid in the peritoneal cavity

**Etiology.** The fluid may be a transudate (*hydroperitoneum*), an exudate (*seroperitoneum*), blood stained (*hæmoperitoneum*) or fatty (*chyloperitoneum*).

*Hydroperitoneum* may result from heart failure, constrictive pericarditis or obstruction of the inferior vena cava. Cirrhosis of the liver. Obstruction of the portal vein, as by pressure from enlarged glands in the portal fissure which may be due to tuberculosis, carcinoma or Hodgkin's disease or the pressure of an aneurysm or thrombosis. Nephritis, pernicious anæmia or leukaemia. *Seroperitoneum* may be due to chronic peritonitis as in tuberculosis, carcinoma or hydatid infection or to polyserositis, or ovarian tumours. *Hæmoperitoneum* may occur in peritonitis associated with tuberculosis or carcinoma. *Chyloperitoneum* may be due to obstruction of or injury to the lacteals or to nephritis.

Ascites is thus a sign of diverse pathological states.

**Pathology.** The fluid. In *hydroperitoneum* the transudate is clear, straw coloured, with a specific gravity usually below 1.015, containing protein under 2% and a few cells. In *seroperitoneum* the fluid may be darker, the specific gravity is usually over 1.015, the protein content is higher (over 3%) and the cells are more numerous. In *hæmoperitoneum* red blood corpuscles are present. *Chyloperitoneum* may be chylous fluid, whitish yellow and turbid with chyle, a pseudo-chylous fluid may form in which the fat comes from degenerating cells.

**Clinical Findings.** The clinical picture varies according to whether the cause is cardiac, hepatic, renal, etc. The local symptoms and signs are as follows. The patient complains of abdominal swelling, heaviness or discomfort. There may also be dyspnoea or palpitations with swelling of the legs.

**On Examination:** The abdomen appears fairly uniformly distended when the patient is lying, with some prominence in the flanks, provided the fluid is not loculated. If there is a large quantity of fluid the umbilicus may be everted and striæ atrophicæ may be seen. Enlarged veins may be present around the umbilicus (*caput Medusæ*) and with portal thrombosis dilated veins appear in the hypogastrium, in which the direction of flow is reversed (i.e., the blood passing from below upwards). On palpation a fluid thrill may be obtained. The thrill is transmitted to one hand placed on the abdomen in the flank, when the other flank is flicked with the finger; the ulnar margin of an assistant's hand should be used to compress the abdominal wall in the centre, to prevent transmission of the thrill along the wall. On percussion there is dulness in the flanks and hypogastrium, and this shifts when the patient turns on his side, the upper portion becoming resonant. If the liver is enlarged it may be felt by suddenly pressing with the fingers over it ("dipping"). With small degrees of ascites the dulness may become apparent in the umbilical region by percussing the abdomen with the patient in the knee-elbow position. Loculated ascites may cause dulness in one zone of the abdomen, which does not shift with change of position. There may be œdema of the legs and scrotum, and the urine may contain a trace of albumin.

**Differential Diagnosis.** Ascites must be distinguished from abdominal distention due to a full bladder, an ovarian cyst, a tumour or flatulent intestinal distention. The bladder should be emptied with a catheter. With an ovarian cyst the swelling is mainly below the navel, the dulness does not extend to the flank or shift as it does with free fluid in the peritoneum, and on vaginal examination displacement of the uterus may be felt. With a tumour, the mass, if palpable, is generally firm and there is no shifting dulness. With flatulent distention the abdomen is hyperresonant, but flatulent distention and ascites often coexist.

• **Course and Complications.** These must vary with the underlying cause of the ascites.

**Prognosis.** Ascites is always a serious condition, in many instances being the harbinger of death. The outlook is more favourable in cases due to congestive heart failure.

**Treatment.** The fluid intake should be limited to 20 or 30 oz. a day, and an endeavour made to increase the urinary output by the use of such measures as Guy's pill (pil. digital. co. B.P.C.) 1 t.d.s. for 3 days, or theophyll. et sod. acetat. gr. 2 in a cup of tea t.d.s. for 3 days. Salyrgan (mersalylum. B.P.) or Novurit can also be used, as described on p. 228. If a favourable result ensues there will be profuse diuresis and the ascites disappears. The diet should be a salt-poor one (see p. 435). If these measures fail, the fluid can be removed by paracentesis, the abdomen being tapped, after preliminary anæsthetisation with 2% solution of Novocain (procain. hydrochlor. B.P.), using a trocar and cannula. The site should be in the mid-line, half-way between the umbilicus and the pubes, or in one flank. The bladder should always be emptied with a catheter before the cannula is inserted.

## THE MESENTERY

The following affections of the mesentery will be briefly described. Inflammation (mesenteritis), structural abnormalities, hæmorrhage, thrombosis, embolus, cysts, new growths, and tuberculous glands.

### Inflammation

#### (*Mesenteritis*)

This may form part of a general peritonitis or be localised to a part of the mesentery as the result of spread of infection from adjacent intestine. Chronic mesenteritis may be associated with visceroptosis (see p. 64).

### Structural Abnormalities

Excessive length of the mesentery may lead to volvulus, and the bowel may become incarcerated through an opening in the mesentery.

### Hæmorrhage

This may occur rarely in the hæmorrhagic varieties of the acute specific fevers, such as small pox, or as the result of degenerative changes such as arteriosclerosis or aneurysm of the mesenteric arteries. It produces symptoms resembling those of intestinal obstruction.

### Thrombosis and Embolus

Venous thrombosis may result from cirrhosis of the liver and congestive heart failure, or develop in association with pyelephlebitis or intestinal or appendicular suppuration or after splenectomy. It may also occur as a manifestation of thrombophlebitis migrans. An embolus may lodge in the mesenteric arteries in cases of infective endocarditis and mitral stenosis. The clinical picture is that of an acute abdominal catastrophe. The patient is suddenly taken ill with severe abdominal pain, collapse, sweating, pallor, low temperature, and frequent pulse. The abdominal wall is not usually rigid. Some blood and mucus are passed by the intestine, in a few hours the abdomen becomes distended, and vomiting and perhaps hæmatemesis set in. Gangrene or perforation of the intestine may occur. The outlook is very grave, and the treatment is surgical.

### Cysts and New Growths

Mesenteric cysts may be hæmorrhagic, serous, lymphatic or due to hydatid infection or to a dermoid. They form rounded swellings in the abdomen and cause vomiting, colic or intestinal obstruction.

New growths are usually secondary malignant deposits.

### Tuberculous Glands

#### (*Tabes Mesenterica*)

*Tabes mesenterica* is described on p. 101 (chronic tuberculous peritonitis).

## CHAPTER II

### THE RESPIRATORY SYSTEM

**Introductory.** Clinical investigations in diseases of the lungs include a routine examination of the chest by inspection, palpation, percussion and auscultation. The lungs should be examined radiologically with a screen and film, and in some cases after the injection of Lipiodol or after establishing an artificial pneumothorax. Tomography may be of value in demonstrating deep-seated cavities, for by this process serial views of the lungs are obtained at varying depths from the surface. The weight of the patient should be charted weekly and a note made of his highest known weight.

The vital capacity of the lungs is estimated by a spirometer. It is the amount of air which can be expired, after a deep inspiration. The average amount for an adult is 3,600 c.c. It is especially diminished in emphysema and is also found to vary with the degree of activity in pulmonary tuberculosis. The sputum should be examined for colour, consistency and odour, and the volume in 24 hours recorded on the temperature chart. Laboratory tests include a search for tubercle bacilli, the predominating organisms, and special tests for spirochaetes, ova of *paragonimus westermani*, streptothricæ, elastic and lung tissue. It may be necessary to determine the Wassermann reaction of the blood, the rate of sedimentation of the red cells and the cell count.

### THE UPPER RESPIRATORY TRACT

#### Hay Fever

(*Allergic coryza. Pollinosis*)

**Definition.** Paroxysmal and seasonal inflammation of the conjunctivæ and nasal mucous membrane.

**Etiology.** Hay fever is due to sensitiveness to certain pollens. In England the pollen is usually that of the "Timothy grass," and more rarely tree pollens. In the autumn the Michaelmas daisy is an occasional cause.

**Pathology.** The condition is one of allergy, the patient exhibiting an abnormal sensitivity to foreign proteins or to altered tissue proteins. There is hyperæmia of the conjunctivæ, of the nasal mucous membrane, and sometimes of the larynx and trachea.

**Clinical Findings.** The patient is usually a young adult, who gives a history that every year in the early summer from May to July he suffers from attacks of sneezing, watering of the eyes and often headache. There may be much watery discharge from the nose and some malaise. He may also suffer from asthma at different periods of the year. The attacks are more common in the country, especially if near a hay field. They also occur in towns.

**Differential Diagnosis.** The diagnosis is established by the seasonal

incidence and the cutaneous reaction of the patient to pollen. Hay fever should be differentiated from paroxysmal sneezing due to local irritation of the nose, or to nervous influences (nervous coryza). In paroxysmal rhinorrhœa, attacks of sneezing or of running from the nose occur at all times of the year. This is probably a vasomotor phenomenon due to allergy, the patient being sensitive to dust, snuff, orris powder, feathers, animal emanations, bacteria, etc.

**Course and Complications.** The course of the disease is limited by the life history of the plants producing the pollen. Asthma or a perennial coryza may develop as complications.

**Prognosis.** This is good, as subsequent attacks may be prevented or modified by appropriate treatment. Hay fever tends to lessen in severity in successive years, and to disappear in about 20 years.

**Treatment. Prophylactic.** Early in the year the cutaneous reaction to Pollaccine should be determined. A control prick is first made through a drop of normal saline with a hypodermic needle. A drop of Pollaccine containing 20 000 units of pollen toxin per mil is placed on the forearm and a prick is made through it. The drops are then wiped off with cotton wool. A positive reaction is shown in about 15 minutes by the formation of an urticarial wheal surrounded by a red areola. Desensitisation should be begun early in February. Subcutaneous injections of Pollaccine are given every other day. The initial dose varies from 40 to 100 units, and a 15% increase is made at each injection. In order to obtain adequate protection a final dose of 50,000 to 100 000 units will be required. A local reaction may be prevented by taking calcium lactate gr. 10 half an hour before the injection. A marked local or general reaction can be checked by the subcutaneous injection of m 3 to 5 of liq. adrenal hydrochlor. Should a reaction occur the next injection should be the largest preceding dose that did not cause a reaction.

**Curative.** A rapid method of desensitisation has been introduced. It is given to patients during the hay fever season and they can be desensitised in about a week. The patient should be in an institution and under skilled supervision during the treatment. The injections are given every 1½ to 2 hours during 14 hours out of the 24 hours. The initial dose is increased by 10% or 20% at each injection, provided no reaction occurs; when the next dose must be smaller. Thus the initial dose of say 100 units may be rapidly raised to 20 000 or more units. Alternatively, considerable relief can often be obtained by the daily subcutaneous injection of 100 units pollen toxin and a subcutaneous injection of m 3 of liq. adrenal hydrochlor. Further treatment for the patient who is suffering from hay fever consists in wearing dark glasses. The nasal mucous membrane may be protected by white vaseline, or by Anesthone cream. Treatment of the nasal mucous membrane by zinc ionisation is not of great value in hay fever. The treatment of paroxysmal rhinorrhœa due to dusts, etc., is similar to that for asthma (see p. 133). Some cases respond satisfactorily to injections of an autogenous vaccine prepared from organisms from the nose.

## Epistaxis

**Definition.** Bleeding from the nose.

**Etiology.** Epistaxis may be due to local or general causes. *Local causes:* Trauma, picking the nose, foreign bodies in the nose, nasal diphtheria, new growths such as a polypus or malignant tumour, lupus, syphilis, telangiectases (which may be hereditary), a fractured base or a pituitary tumour. *General causes:* Epistaxis may be associated with high blood pressure, mitral stenosis, portal cirrhosis of the liver, enteric fever, whooping-cough, blood diseases such as leukæmia, anæmia, hæmophilia and purpura, and a lowered atmospheric pressure such as occurs at high altitudes. It may also occur at puberty without any discoverable cause.

**Pathology.** The bleeding point is often situated in Kiesselbach's area, at the anterior and inferior part of the septum nasi.

**Differential Diagnosis.** Blood which passes backwards from the nose may be coughed up and raise the question of hæmoptysis. A careful naso-pharyngeal examination will usually reveal the bleeding point. In other cases the blood may be swallowed and vomited or cause melæna.

**Course and Complications.** Usually the bleeding stops in a short time, but it may be persistent and cause anæmia and debility.

**Prognosis.** This depends on the cause. Epistaxis may be trivial, or it may be a symptom of a fatal disease.

**Treatment.** This varies with the cause. In children the bleeding usually stops without treatment, unless due to a foreign body, which should be removed. In other cases a cold compress should be applied to the nose with the patient lying down, or a little cotton-wool inserted into the nostril and pressure applied over it. The wool may be soaked in liq. adrenal. hydrochlor. or in viper venom as described on p. 522. In persistent cases the bleeding point should be sealed by the galvanocautery at cherry-red heat. Epistaxis associated with a high blood pressure is a useful safety valve and does not require to be checked, unless very excessive.

## THE LARYNX

## Acute Simple Laryngitis

**Definition.** Acute catarrhal inflammation of the larynx.

**Etiology.** Acute simple laryngitis may be a symptom of a common cold, or result from inhalation of irritants. It may also occur as an early manifestation of measles, or be due to improper production or over-use of the voice.

**Pathology.** There is hyperæmia of the vocal cords and epiglottis, with exudation of mucus.

**Clinical Findings.** The patient complains of alteration in his voice, varying from harshness to hoarseness or aphonia. The throat may feel raw and there may be malaise with an irritating cough.

**On Examination:** The temperature may be a little above normal, and laryngoscopic examination will reveal the redness or swelling of the larynx.

**Differential Diagnosis** Acute simple laryngitis must be differentiated from functional aphonia and from such serious conditions as laryngeal diphtheria, tuberculosis, syphilis or carcinoma of the larynx, which are described later

**Course and Complications** The laryngitis usually improves in 7 to 10 days but the course is more prolonged if the voice has been strained or if irritants have been inhaled

**Treatment.** The voice should be rested. If there is any fever the patient should be kept in bed, the temperature of the room maintained at 65° F and the air moistened by a steam kettle. A steam inhalation should be used for 5 minutes 3 times a day such as *Ol abietis m 10 mag carb lev gr 10, aquam ad m 120 M 120 in 1 pint of steaming water at 165° F*. Hot compresses applied to the neck are often comforting. Cough can be checked by a sedative mixture such as *Tnc opu camph m 10 tnc ipecac m 2 syr pruni scroto m 30, aq chlorof ad fl oz. ½ Fl oz ½ f d s*

### Chronic Simple Laryngitis

**Definition** Chronic catarrhal inflammation of the larynx

**Etiology** Chronic simple laryngitis may result from irritants, such as tobacco and various forms of dust, from over use or faulty use of the voice, or from prolonged coughing associated with chronic bronchitis or pulmonary tuberculosis. It may also be secondary to chronic infection in the mouth, nose or pharynx

**Clinical Findings** The onset is usually insidious, the patient complaining of hoarseness or of weakness of the voice. The throat may feel dry and there may be an irritating cough

**On Examination** The pharynx is usually red and swelling or redness of the vocal cords may be seen

**Treatment.** The patient should rest the voice as much as possible, and give up smoking and alcohol. A steam inhalation should be used at night, as described above and during the day a potassium chlorate lozenge may be sucked occasionally. Subsequently lessons should be taken in voice production, if the laryngitis has been caused by errors in this respect, and a dusty occupation should be changed, if possible. In obstinate cases a change of air and rest may effect a cure

### Tuberculous Laryngitis

**Etiology** Tuberculous laryngitis is usually associated with pulmonary tuberculosis and is due to infection with the tubercle bacillus

**Pathology** The disease may be localised to the vocal cords, interarytenoid space, ventricular bands or epiglottis. There may be swelling, infiltration or ulceration. The posterior part of the vocal cords is most often affected

**Clinical Findings** The patient's first complaint may be of hoarseness or of loss of voice. In other cases he may develop these symptoms as a complication of a recognised pulmonary tuberculosis. If the epiglottis is involved the patient may notice difficulty in swallowing liquid foods, any attempt to do so provoking cough. In the later stages there may be



severe pain on swallowing, felt in the throat and radiating to the ear.

*On Examination:* Various lesions may be found in the larynx, such as swelling or ulceration of the inter-arytenoid space or of a vocal cord. The vocal cord may be fixed by the granulation tissue.

*Differential Diagnosis.* Tuberculous laryngitis has to be differentiated from a simple laryngitis which may occur in pulmonary tuberculosis, and from syphilis or a new growth. The opinion of an expert laryngologist will be required. In addition the sputum must be tested for tubercle bacilli, the lungs X-rayed, the Wassermann reaction determined, and a portion of the diseased tissue may require microscopical examination after removal by punch forceps.

*Course and Complications.* Tuberculous laryngitis may be cured by suitable treatment, especially in the early stages; often it is, however, a progressive lesion. Pulmonary tuberculosis is usually present.

*Treatment.* This is described on p. 158.

### Syphilis of the Larynx

*Etiology.* Laryngeal syphilis is due to infection with the *Treponema pallidum*.

*Pathology. Congenital syphilis:* A catarrhal syphilitic laryngitis may occur in infancy, or a gumma may develop about the age of puberty.

*Acquired syphilis:* Lesions may occur during the secondary stage, such as patchy hyperæmia of the cords, with formation of mucous spots. In tertiary syphilis, the lesions include infiltration, serpiginous ulceration, gumma formation or stenosis of the larynx. The anterior part of the larynx is more commonly affected than the posterior.

*Clinical Findings.* There is usually no pain and no cough, but the voice is hoarse or raucous. Stridor is indicative of stenosis, due to contraction of fibrous tissue.

*Differential Diagnosis.* This is as for tuberculous laryngitis. The diagnosis is established by the laryngoscopic appearances, the Wassermann reaction and the response to treatment.

*Treatment.* A full course of anti-syphilitic treatment is required (see p. 570). Caution must be exercised in the administration of iodides, as œdema of the larynx may ensue.

### Tumours of the Larynx

These may be simple or malignant.

#### Simple Tumours

These include: Papilloma, fibroma, "singer's nodule" (keratoderma), angioma and degeneration cysts.

*Pathology.* Innocent tumours often grow from the vocal cords, at the junction of the anterior and middle thirds of the cord. A papilloma is frequently pedunculated.

*Clinical Findings.* The patient may have no symptoms, but if the tumour is growing from a vocal cord there is usually hoarseness, and there may be some stridor. An angioma may give rise to hæmoptysis.

*Differential Diagnosis.* This is made by laryngoscopic examination and the microscopical appearances of the tumour, after removal.

*Treatment.* This is surgical.

### Malignant Tumours

**Pathology** Intrinsic growths occur in the larynx. The commonest variety is the epithelioma. This may be a sessile tumour on a vocal cord, or an infiltration of the tissues of the cord. It may occur in other sites, such as the ventricular band or the posterior commissure. Extrinsic growths may be found in the epiglottis or around the cricoid cartilage. Secondary deposits in the cervical glands occur comparatively late in cases of intrinsic laryngeal carcinoma, but early in extrinsic growths. Spheroidal celled carcinoma and sarcoma are both rarely seen.

**Clinical Findings** The patient is usually a male over the age of 40. He first complains of hoarseness or of weakness of the voice, often thought to be due to a cold, but which persists despite treatment. Later there is pain, especially on swallowing, and progressive deterioration of health, due to cachexia from the growth.

**On Examination** The growth is seen on laryngoscopic examination. Later, secondary deposits occur in the cervical glands, and secondary infection of the growth may give rise to cervical cellulitis or abscess formation.

**Differential Diagnosis** This can only be carried out by an expert laryngologist. The difficulties are well known, owing to the prominence given to the fatal illness of the late Kaiser Friedrich III.

**Treatment** This is surgical, or by X rays or radium.

### Œdema of the Larynx

**Definition** Swelling of the larynx, due to exudation of fluid.

**Etiology** Œdema of the larynx may be inflammatory or non-inflammatory. *Inflammatory œdema* This may occur in association with acute catarrhal laryngitis, septic pharyngitis, cellulitis of the neck (Ludwig's angina), erysipelas of the face and neck, ulceration of the larynx due to syphilis or tuberculosis, or rarely as a complication of diphtheria, scarlet fever, typhoid fever, etc.

*Non-inflammatory œdema* This may be a manifestation of angio-neurotic œdema, or be associated with cardiac or renal disease, or it may result from hypersensitiveness to drugs, such as iodides.

**Pathology** The swelling affects chiefly the aryteno-epiglottidean folds, the vocal cords being only slightly involved owing to the tense attachment of their mucous membrane. The epiglottis and subglottic region of the larynx may be very swollen.

**Clinical Findings** The patient is often a child, who is suddenly seized with dyspnoea and symptoms of asphyxiation. There is a hoarse cry and inspiratory stridor. The swollen epiglottis can usually be seen on oral examination.

**Treatment.** If the swelling is due to angio-neurotic œdema, a subcutaneous injection of m 7 of liq. adrenal hydrochlor. should be given immediately, and an adrenaline spray may be used in an atomiser containing liq. adrenal hydrochlor. 1 part, and normal saline 1½ parts. If the œdema is due to other causes, the immediate treatment consists in giving ice to suck, the application of ice-cold compresses to the neck, and scarification of the œdematous epiglottis with a curved

guarded bistoury, after cocainisation. In very severe cases tracheotomy may be necessary. In cases due to iodide idiosyncrasy relief may usually be obtained by giving sod. bicarb. gr. 60 t.d.s., and by omitting the iodides.

### Croup

**Definition.** A crowing form of respiration met with in infants and young children.

**Etiology.** The following varieties may be distinguished :—

1. *Inflammatory*: Laryngitis stridulosa. Membranous laryngitis (diphtheritic and non-diphtheritic). Simple acute laryngitis. Whooping-cough.

2. *Reflex*: Laryngismus stridulus. Croup associated with enlarged tonsils and adenoids, with dentition, tetany and rickets.

3. *Mechanical*: Infantile laryngeal stridor. Laryngeal polypi. Foreign bodies in the larynx. Pressure of an enlarged thymus upon the trachea (see p. 122).

Certain of these varieties will be briefly described.

### Laryngitis Stridulosa

**Clinical Findings.** This is an affection of young children. The symptoms of a cold are usually present, with a cough and perhaps some hoarseness during the day. The breathing is easy when the child goes to bed, but he wakes up during the night with an attack of "croup," characterised by cough, inspiratory stridor and cyanosis. This usually passes off in from a few minutes to half an hour, when the child falls to sleep again.

**Prognosis.** The condition may be alarming, but is not fatal.

**Treatment.** If the attack does not rapidly subside the child should be put in a hot bath. A steam inhalation from hot, but not boiling, water should also be given (see p. 110) or the nose and pharynx sprayed with neb. adrenal. et ephedrin. (B.P.C.). If these measures do not afford relief, an emetic dose of tnc. ipecac. should be given, such as m. 120 for a child of 6 years. Subsequently if the tonsils and adenoids are enlarged, they should be removed.

### Laryngismus Stridulus

#### (Spasmophilia. Breath-holding Attacks)

**Etiology.** This variety of croup may be a manifestation of tetany (see p. 659). It is considered by some to be analogous to asthma, and is often associated with rickets, enlarged tonsils or dentition.

**Pathology.** There is laryngeal spasm, but no inflammation.

**Clinical Findings.** The patient is usually an infant, who is suddenly seized during the night with an attack of laryngeal spasm. After some struggling the vocal cords relax and the air enters with a crowing sound. Carpo-pedal spasm may be present during the attack (see p. 660).

**Prognosis.** Death may occur during the attack.

**Treatment.** *During the attack:* The tongue should be pulled forward by passing the finger behind it and cold water sprinkled over

the head and chest. If this fails, a little chloroform placed over the mouth on lint may cause the child to inhale. *After the attack* The blood calcium should be estimated, and if low, calcium lactate given by mouth in doses of gr 1 t d s to a child of 1 year. Rickets should be treated if present (see p 621), and enlarged tonsils and adenoids should be removed.

### Infantile Laryngeal Stridor

This is due to a congenital deformity of the larynx, in which the orifice is unduly small. It disappears as the child grows.

### Laryngeal Paralysis

**Introductory** The vocal cords are adducted on phonation, and abducted with inspiration. These movements are effected by intrinsic muscles. The adductor group includes the lateral crico arytenoid muscles, the inter arytenoid and the outer part of the thyro arytenoid muscles. The abductors are the posterior crico arytenoid muscles. The crico thyroid muscles also help to render the cords taut.

Adduction is a specialised movement, controlled by a cortical centre. Abduction is mechanical, and regulated by a centre in the medulla. In functional paralysis adduction is affected, whereas in organic disease abduction is first lost. *Semon's law* states that in a progressive organic lesion the intrinsic laryngeal muscles are affected in the following order: Abductors, tensors and adductors.

The nervous path consists of a centre in the third frontal convolution of the brain, and this is bilateral. Thence upper motor neurones pass in the internal capsule to the lower motor neurone centre in the medulla (X and XI cranial nerve nuclei). The lower motor neurones run in the vagus, the superior laryngeal branch of which supplies the crico thyroid muscle, and the recurrent laryngeal branch innervates the other intrinsic muscles (the lateral and posterior crico arytenoids, the inter arytenoid and the thyro arytenoids).

### Functional Aphonia

**Etiology** Functional aphonia occurs in association with hysteria, shell shock, debility, or at times with pulmonary tuberculosis.

**Clinical Findings.** The patient speaks in a whisper, but can cough normally. There is no dyspnoea.

**Laryngoscopic Examination.** The cords appear normal at rest, but on phonation adduction is incomplete. Abduction is normal with inspiration.

**Treatment.** The general condition of the patient should be improved with a tonic such as syrup of glycerophosphate (B.P.C.) in 60 t d s. The voice may sometimes be immediately restored by local intralaryngeal faradic stimulation, or by firm depression of the tongue with the middle finger of the right hand, while the patient is told to cough and finish the cough on the sound "Ah". Suggestion is of great value in some cases. The aphonia, however, often recurs.

## Organic Laryngeal Paralysis

**Etiology.** The lesion may be in the brain, medulla, vagus, superior laryngeal or recurrent laryngeal nerve, or there may be a local laryngeal lesion, such as ankylosis of the arytenoid cartilage. *A cerebral lesion:* This must be bilateral to paralyse the vocal cords, for stimulation of one centre causes adduction of both cords. It is therefore very rare. *A nuclear lesion:* Involvement of the nuclei of the X and XI cranial nerves results in homolateral cord paralysis. The chief causes are tabes dorsalis, a gumma, a hæmorrhage, a new growth, disseminated sclerosis, syringomyelia, amyotrophic lateral sclerosis, and labio-glossopharyngeal paralysis. These lesions are often bilateral. *The vagus:* This may be involved in a fracture or tumour of the base of the skull or by pachymeningitis or neuritis. *The superior laryngeal nerve:* This is rarely affected, but it may be compressed by enlarged cervical glands, or damaged by trauma or by diphtheritic neuritis. Such a lesion causes loss of tension in the vocal cords. *The recurrent laryngeal nerve:* This may be compressed by an aneurysm, glands in the neck, a thyroid tumour, carcinoma of the œsophagus, chronic apical pleurisy especially on the right side, a dilated left auricle in mitral stenosis, a mediastinal tumour, cervical or mediastinal abscess, and a pericardial or pleural effusion. It may also be affected by neuritis due to such causes as cold, diphtheria, alcohol, arsenic or lead. It may be injured at an operation. The following varieties of organic paralysis may be due to any of the causes given above.

## Unilateral Abductor Paralysis

**Clinical Findings.** There is no cough, the voice is normal or hoarse, and there may be some dyspnœa on exertion.

**Laryngoscopic Examination:** The cord lies near the mid-line at rest, it does not abduct on inspiration, but on phonation the cords meet.

## Total Paralysis of One Cord

**Clinical Findings.** The voice may be low and hoarse, but there is no cough and usually no dyspnœa.

**Laryngoscopic Examination:** The cord is in the "cadaveric" position, mid-way between adduction and abduction. It does not move on phonation or on inspiration. On phonation the sound cord comes across the mid-line to meet the paralysed one.

## Bilateral Abductor Paralysis

**Clinical Findings.** The voice is practically normal, but there is inspiratory stridor.

**Laryngoscopic Examination:** Both cords at rest lie near the mid-line. They adduct on phonation, but there is no abduction on inspiration. Tracheotomy may be required to relieve the dyspnœa.

## Bilateral Complete Paralysis

**Clinical Findings.** The patient can only whisper, but stridor is not present.

*Laryngoscopic Examination* Both cords are in the "cadaveric" position, and they are immobile on phonation and on inspiration

## THE TRACHEA

### Tracheitis

*Etiology* Tracheitis may be acute or chronic *Acute Tracheitis* This is usually bacterial in origin, occurring either with a cold, which extends to the large bronchi, or in association with whooping cough or influenza. It may also be met with in measles, diphtheria or enteric fever. Inhalation of irritants, such as poison gases or steam, is a causative factor in some cases. *Chronic Tracheitis* This may follow acute tracheitis, or be due to chronic irritation from smoking. It may be secondary to chronic inflammation of the nose or larynx, or to a local lesion in the trachea, such as a tumour or gumma.

*Pathology* The inflammatory changes vary in degree from the vascular engorgement exemplified by the "pink" trachea of influenza, to membrane formation with sloughing as may occur in diphtheria or gas poisoning.

*Clinical Findings* In acute tracheitis the patient complains of a sense of soreness under the sternum, but the symptoms of the associated laryngitis and bronchitis are usually more noticeable.

*Treatment.* In the acute stages the patient should be kept in a warm room, and if there is fever he should be in bed. Counter irritants should be placed over the upper part of the sternum and lower part of the neck, such as camphorated oil (*lin camphoræ B.P.*). A sedative cough mixture should be given, such as Tne opii camph m 10, liq ammon acetat m 60, syrup pruni serotin m 30, aq chlorof ad fl oz  $\frac{1}{2}$ . Fl oz  $\frac{1}{2}$  t d s. A steam inhalation containing tne benzoin. co m 60 in a pint of steaming water, at a temperature of 165° F, should be used 2 or 3 times a day.

### Tracheal Obstruction

*Etiology* The obstruction may be due to causes in the lumen of the trachea, in the wall or outside the wall.

1 *In the lumen* This may result from an inhaled foreign body, or from a pedunculated tumour, such as a papilloma.

2 *In the wall* Obstruction may be caused by cicatrization of a wound or tracheotomy scar, or by fibrosis following the inhalation of severe irritants. Syphilis, leprosy, scleroma and secondary malignant deposits are rare causes of obstruction.

3 *Outside the wall* Obstruction may result from pressure due to an enlarged thyroid ("scabbard" trachea), enlarged cervical glands, especially if affected by malignant growths or Hodgkin's disease, an aneurysm, an enlarged thymus or a mediastinal tumour.

*Clinical Findings* These vary with the degree of obstruction and the suddenness of its onset. The most noticeable symptoms are dyspnoea and tracheal stridor. Pressure from an enlarged thymus in infants, resulting in tracheal stridor, is very liable to be mistaken for croup.

due to laryngeal obstruction. The dyspnoea is often relieved by leaning the infant forward. The temperature is normal, and the cyanosis and dyspnoea may improve markedly from time to time. A foreign body which has passed through the larynx often gives rise to very slight symptoms when it is in the trachea, and it usually rapidly passes into a bronchus. In slowly developing obstruction the stridor is often heard first when the patient is asleep. When the obstruction is severe, respiratory excursions of the larynx are obvious, and the accessory respiratory muscles are in action.

**Treatment.** *This varies with the cause of the obstruction.* Tracheotomy must not be performed in cases of thymic stridor, as it will only precipitate death. The possibility of X-ray treatment should be considered, and the baby must not be allowed to lie down. Foreign bodies in some cases can be located by X-ray examination and removed with a bronchoscope. In organic stenosis in the cervical portion of the trachea, it may be possible to relieve the dyspnoea by a low tracheotomy. Usually nothing can be done to relieve obstruction in the mediastinum due to external pressure, unless there is a tumour which can be removed by operation.

### Tracheal Diverticula

Tracheal widening, with protrusion of its mucous membrane between the rings, is a rare condition, revealed at times by endoscopy or by Lipiodol examination.

## THE BRONCHI

### Acute Bronchitis

The following varieties are described: Catarrhal, capillary, suppurative, and fibrinous.

### Acute Catarrhal Bronchitis

**Definition.** Acute inflammation of the larger bronchial tubes, usually associated with tracheitis.

**Etiology.** *Primary cases:* The exciting organisms may be the *Neisseria catarrhalis*, the *Diplococcus pneumoniae* (pneumococcus), the *Bacterium friedländeri* (pneumobacillus), staphylococci, streptococci, etc. Inhalation of dust and chemical irritants may also cause acute bronchitis. *Predisposing causes:* 1. Age: Childhood and late adult life. 2. Sex: Males predominate. 3. Climate: Damp and wet. 4. Season: Autumn and winter. 5. Heredity: There is often a hereditary factor.

*Secondary cases:* Acute catarrhal bronchitis may occur as a complication in such diseases as measles, whooping-cough, influenza, enteric fever, diphtheria, nephritis, pulmonary tuberculosis, malaria, etc.

**Pathology.** The lower part of the trachea and the main bronchi are affected. The mucous membrane is hyperæmic in the early stages, with little secretion; later a thin mucous exudate appears which becomes muco-purulent, and finally ceases.

**Clinical Findings** The patient is taken ill with a cold, which passes down to the chest. There is malaise, with perhaps headache and shivering as the temperature rises. A sense of rawness may be felt under the sternum or in the second and third intercostal spaces near the sternum. A dry cough causes pain in the chest, which disappears as the secretion forms and is expectorated.

**On Examination** The temperature may be raised to 100° or 101° F, the respiration may be slightly increased to 18 or 20, and the pulse is also somewhat frequent, 90 to 100. The chest. **Inspection** Movement is good and equal. **Palpation** Rhonchal fremitus may be present on both sides, tactile fremitus is normal. **Percussion** The note is normal. **Auscultation** The breath sounds are harsh or they may be almost obscured by sonorous or sibilant rhonchi. As the secretion loosens, bubbling rales may be heard. Vocal resonance is normal. The sputum is scanty and tenacious at first, later it increases and is more purulent.

**Differential Diagnosis** There is usually no difficulty in the diagnosis, either of the primary or secondary cases. The sputum should be examined and the lungs X rayed to exclude the possibility of tuberculosis.

**Course and Complications** The disease usually pursues a course lasting 2 to 3 weeks, the temperature falling to normal in a week or so. Chronic bronchitis may ensue as a sequela.

**Prognosis** This is good, unless the patient is very young or very old, when the disease may prove fatal.

**Treatment.** The patient should be kept in bed in a warm and moist atmosphere the temperature being maintained between 60° and 65° F day and night, and a steam kettle used. The chest should be rubbed daily with a liniment such as camphorated oil (lin camphoræ B P) and covered with a gamgee jacket. During the dry stage a steam inhalation may be used such as *Ol abietis* m 10 *mag carb lev gr* 10, *aq ad m* 120. *M* 120 in a pint of steaming water at 105° F. The vapour to be inhaled from a Nelson's inhaler for five minutes night and morning. The following cough medicine may be given during this stage, *Vin antimon (B P C)* m 3 *tnc ipecac m* 5 *liq ammon acetat.* m 120 *syr tolu m* 30 *aq ad fl oz* 4 *fl oz* 4 t.d.s. Sleep may be induced by the use of some preparation such as Sedobrol 1 or 2 tablets in a cup of hot water or in some adult cases *pulv ipecac et opii gr* 10. The bowels should be opened daily with a saline such as *mag sulph gr* 60 to 1.0 mane. During the febrile stage the diet should be liquid or semi solid. As the secretion loosens and the cough becomes easier, a stimulant expectorant should be given, such as *Ammon carb gr* 3, *tnc scilla m* 5 *sp chlorof m* 7 *infus senega rec. ad fl oz* 1 *fl oz* 1 *ex aqua t.d.s.* If there are signs of dilatation of the right side of the heart cardiac and respiratory stimulants should be given, such as hypodermic injections of digitalin gr 1/100 and strychnin hydrochlor gr 1/60 four hourly or Coramine (nikethamidum B P Add) 1.5 ml six hourly. If possible a period of convalescence should be arranged for, in a warmer climate.



### Capillary Bronchitis

This variety of acute bronchitis is indistinguishable from broncho-pneumonia (see p. 145).

### Acute Suppurative Bronchitis

#### (*Acute Purulent Bronchitis. Suffocative Catarrh*)

**Definition.** An acute variety of bronchitis, occurring at times in epidemics, and characterised by profuse purulent expectoration.

**Etiology.** The *Diplococcus pneumoniae*, the *Hæmophilus influenzae* and the *Neisseria catarrhalis* were the organisms frequently found in the sputum during the epidemic in 1916-17.

**Pathology.** There is a diffuse purulent inflammation of the medium and smaller-sized bronchi. The alveoli may contain a fibrinous exudate.

**Clinical Findings.** The patient is suddenly taken ill with malaise, shivering and high fever. He develops a cough, is very short of breath and brings up thick sputum.

**On Examination:** The patient is very cyanosed and obviously dyspnoeic. The *alæ nasi* and other accessory muscles of respiration are in action. The temperature may be raised to 104° F. and the pulse and respirations are rapid. The lungs: There is usually no dulness and tactile fremitus is present over both lungs. The breath sounds are rather weak and obscured by medium bubbling râles from apex to base. As much as 10 to 15 oz. of purulent sputum may be expectorated in 24 hours.

**Differential Diagnosis.** The intense cyanosis, dyspnoea and purulent sputum serve to establish the diagnosis from other varieties of acute bronchitis.

**Course and Complications.** These vary with the severity of the disease; recovery may occur in 2 or 3 weeks or death in 2 or 3 days. Heart failure is an important complication.

**Prognosis.** This is always very grave.

**Treatment.** This is in the main as for acute catarrhal bronchitis, but the cyanosis should be relieved as far as possible with oxygen administered continuously through a double nasal tube or the B.L.B. mask (see p. 143).

### Acute Fibrinous Bronchitis

#### (*Acute Plastic Bronchitis*)

**Definition.** Acute inflammation of the bronchi, with cast formation.

**Etiology.** The cause is unknown, but pneumococci or streptococci may be present in the casts.

**Pathology.** The casts of the bronchial tree may be solid or hollow. They are greyish in colour and composed of fibrin and mucin. The formation of the cast is preceded by an acute inflammation of the bronchial mucous membrane.

**Clinical Findings.** The onset is usually sudden with malaise, fever, cough and shortness of breath. There may be pain on one side of the chest, in relation to the site of the formation of the cast.

**On Examination:** In addition to the pyrexia, there may be

cyanosis. The signs in the chest may be very slight, or an area of collapse may be detected in one lung, with some dullness and weak air entry. In some instances a peculiar flapping sound is heard, known as the *bruit de drapeau*, as the air passes by the cast.

**Differential Diagnosis** Other causes of dyspnoea, such as asthma, laryngeal and tracheal obstruction, or bronchial obstruction due to other causes require consideration. In some cases there is a history of similar previous attacks.

**Course and Complications** Usually the cast separates in 2 or 3 days, and is expectorated with immediate relief to the symptoms. The cast may be demonstrated by floating the sputum on water.

**Prognosis** This is good as regards the immediate future, but recurrent attacks are very liable to occur.

**Treatment.** A steam inhalation, as described on p. 116, should be given in the acute stage, and between the attacks a course of pot. iod. gr. 5 to 15 t. i. d. should be taken.

### Chronic Bronchitis

The chief varieties are catarrhal, suppurative, and fibrinous.

#### Chronic Catarrhal Bronchitis

**Definition.** Chronic bronchial inflammation.

**Etiology.** Chronic bronchitis may be a recurrent hibernal sequela of an acute attack of bronchitis, or it may start insidiously, due to infection with catarrhal organisms. In some instances chronic bronchitis is secondary to cardiac or renal disease, or to infection in the naso-pharynx or cranial sinuses. *Predisposing causes* 1 Age. Usually over 40. 2 Sex. Males predominate. 3 Climate. Damp and fog. 4 Season. Late autumn, winter and spring. 5 Habits. Over-indulgence in alcohol and tobacco. 6 Dusty occupations.

**Pathology.** The mucous membrane of the bronchi tends to atrophy, with thickening of the bronchial walls. Emphysema and bronchiectasis may develop.

**Clinical Findings.** The patient is often an adult male over the age of 40 who gives a history of recurrent bronchitis every winter. He complains of cough, shortness of breath, and expectoration.

**On Examination.** There is often slight cyanosis of the face, and the fingers may be clubbed. The chest. *Inspection.* Movement is restricted, especially if there is emphysema. *Palpation.* Rhonchal fremitus may be present and tactile fremitus diminished. *Percussion.* The note is hyperresonant, and the area of cardiac and hepatic dullness is often encroached on by the emphysematous lung. *Auscultation.* The breath sounds are harsh or weak, and expiration is usually prolonged. Scattered rhonchi or râles may be heard. In some cases the adventitious sounds are only audible when the patient lies down, or after some exertion. The vocal resonance is either normal or diminished. Cardiac dilatation may be present. The sputum. This may be scanty, and in the form of sticky masses (the *crachats perlés* of Laennec) in the dry variety of chronic bronchitis (*catarrhe sec*). It is

abundant, resembling unboiled white of egg and water, in the variety of bronchitis known as *bronchorrhœa serosa* or *pituitous catarrh*. In the majority of cases there is a small quantity of greenish-black, rather thick sputum and streaks of blood may be present from time to time.

**Differential Diagnosis.** This usually presents no difficulty. Tuberculosis should be excluded by an X-ray and sputum test, and a bronchogram may be required in doubtful cases of bronchiectasis. Early hypertensive left-sided cardiac failure may be mistaken for chronic bronchitis.

**Course and Complications.** The course is progressive, with seasonal intensification. In the early stages the bronchitis is only present in the winter; later it persists, to a milder degree, through the summer. Complications include emphysema, asthma, bronchiectasis and dilatation of the heart.

**Prognosis.** This is usually unfavourable, unless the course of the disease can be modified by climatic treatment.

**Treatment.** This is largely climatic. If possible, the patient should winter abroad, out of Europe, as in Egypt or South Africa. Any septic focus in the naso-pharynx should be removed. In England an outdoor occupation, or one involving the inhalation of smoke or dust, is unsuitable. An autogenous vaccine begun during September may be helpful. The initial dose should be small, such as 50,000 of the predominating organism. The vaccine should be given once a week and the dose not increased to more than 1 million. Much better results are obtained with these small doses than with the larger ones usually employed. If the sputum is difficult to bring up, the hot-water medicine is helpful. This contains Sod. chlorid. gr. 3, sod. bicarb. gr. 5, sp. chlorof. m. 5, and aq. anisi dest. (B. P. C.) ad fl. oz. 1. Fl. oz. 1 in 1 fl. oz. of hot water on rising, and repeated during the day if required. If there is a looser cough an alkaline iodide mixture is helpful, such as Ammon. carb. gr. 5, pot. iod. gr. 3, pot. bicarb. gr. 15, sp. chlorof. m. 5, aquam ad fl. oz. 1. Fl. oz. 1 t.d.s.

### Chronic Suppurative Bronchitis

In this variety of chronic bronchitis the bronchial secretion is retained for a time in some of the bronchi and becomes offensive. It may give rise to periodical unexplained rises of temperature, and resemble the early stages of bronchiectasis. Creosote should be given as for bronchiectasis (see p. 130).

### Chronic Fibrinous Bronchitis

The patient, who is a sufferer from chronic bronchitis, may from time to time have an attack of dyspnoea or a sense of constriction in the chest, which is relieved by the expectoration of a fibrinous bronchial cast. A course of iodides should be given as described above.

### Bronchiectasis

**Definition.** Dilatation of the bronchi.

**Etiology.** There are three main etiological factors: 1. *Mechanical*.

Collapse of a bronchus, either by partial obstruction from within or by pressure from without, causes a fall of the intrapleural pressure with secondary dilatation of the collapsed bronchus, resulting in bronchiectasis. The causes include inhalation of a foreign body such as a bone, a tooth, a portion of a bath sponge, a fragment of tonsil or adenoid tissue removed at an operation, or plugs of mucus. Pressure of an aneurysm or tumour may also cause collapse. Pulmonary fibrosis associated with pleural adhesions may by traction lead to bronchial dilatation. This may follow pneumonia or bronchopneumonia, syphilis or tuberculosis of the lungs, or be secondary to chronic pleurisy or penetrating chest wounds.

**2 Infective** The bronchial wall may be weakened by chronic intrabronchial suppuration, and this, together with a persistent cough, may lead to bronchiectasis. It may thus be associated with the inhalation of septic matter into the bronchi with chronic suppurative bronchitis and with lung abscess. Factors 1 and 2 are often combined.

**3 Congenital** This is an error of development.

**Pathology** The bronchial dilatation may be 1 *Cylindrical, tubular or rat tail*. This dilatation is uniform or narrower at its termination. 2 *Fusiform or glove finger*. The dilatation is wider at its termination. This occurs especially in a collapsed lung. 3 *Saccular or globular*. This resembles a bunch of grapes. 4 *Moniliform or bead-like*. This suggests strung beads. The left lower lobe is most often affected. Acute bronchiolectasis, in which the bronchioles are dilated, giving the lung a honeycomb appearance, may occur in young children or in adults after an illness such as influenza.

**Clinical Findings** The patient is usually a child or a young adult, although bronchiectasis may exist in elderly patients who are considered to be suffering from chronic bronchitis. There may be a history of some antecedent causative condition such as pneumonia or bronchopneumonia. The patient notices first that he has a cough, and the sputum may contain small amounts of offensive material, or it may be noticed that the breath is offensive after coughing, or that the sputum is influenced by change of posture. This probably represents a pre bronchiectatic stage in which a bronchogram may reveal no abnormality. The amount of sputum gradually increases and is more persistently offensive. In other cases periodical unexplained rises of temperature may be the only symptom or there may be recurring hæmoptysis, without any other symptoms (*forme hæmoptique aigue*). In an established case the patient may say that life is unbearable owing to the amount of sputum and its horrible odour.

**On Examination** In an early case The characteristic signs are the offensive nature of the sputum or of the breath, but both of these may be absent. The chest There may be slight dullness at one base with weaker breath sounds and a few persistent rales. In an established case There is usually evidence of toxæmia, as shown by wasting, or stunted growth in a child, cyanosis and clubbing of the fingers and toes. The chest. The signs are those of fibrosis and excavation. Inspection Movement may be less on the affected side. Palpation Vocal fremitus is



FIG. 5. RADIOGRAM AFTER LIPIODOL INJECTION, SHOWING FUSIFORM BRONCHIECTASIS IN A COLLAPSED LOWER LOBE BEHIND THE HEART.

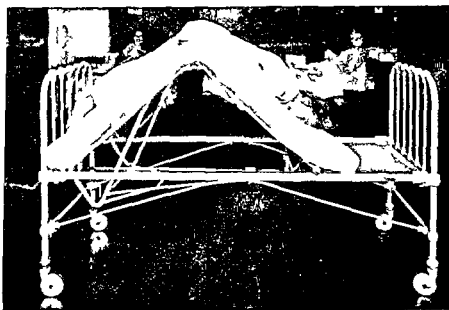


FIG. 6 POSTURAL DRAINAGE ON A NELSON BED

diminished, usually over the affected area. The cardiac apex may be displaced to the side of the lesion. Auscultation: The air entry is usually weak over the bronchiectatic area, and some leathery, creaking or fibroid râles may be heard. If there is a cavity which is comparatively empty and in communication with a bronchus, the breathing may be bronchial or cavernous, with bronchophony and whispering pectoriloquy. Coarse râles may be heard. If the cavity is full there is dulness, with weak breath sounds and diminished voice conduction. The sputum: Expectoration is affected by change of posture. Thus it may come up chiefly on rising in the morning or on lying down at night. It is offensive. The amount may vary from about 1 to 20 oz. or more in the 24 hours. On standing in a conical vessel it may settle into three layers, frothy above, turbid in the centre and a deposit below. In the deposit the evil-smelling Dittrich's plugs may be found. Frequently the sputum is uniformly thick greenish pus. Hæmoptysis may occur from time to time. The temperature chart may show rises corresponding with periods of retention of secretion, and falls when the expectoration is more profuse. A direct X-ray of the lungs may afford some suggestion of dilatation of bronchi, which may be confirmed by tomography, but an X-ray after intratracheal injection of Lipiodol or Neo-Hydriol (ol. iodisat. B.P.Add.) will reveal the outline of the bronchi and thus establish the diagnosis (see Fig. 5).

**Differential Diagnosis.** The diagnosis usually lies between a condition of suppurative bronchitis, interlobar empyema, a lung abscess, congenital cystic disease of the lungs, or pulmonary tuberculosis and various causes of recurrent pyrexia. An apical bronchiectasis cannot be differentiated from tuberculous infiltration by a direct X-ray film. The lipiodol examination enables the diagnosis of bronchiectasis to be made.

**Course and Complications.** The course is usually progressive for a time and then a stationary stage is reached. There is a great tendency for the other lung to become similarly affected. Complications include septicæmia, lardaceous disease, cerebral or spinal cord abscess, empyema, pyopneumothorax, suppurative pericarditis or gangrene of the lung.

**Prognosis.** This is grave unless adequate treatment is secured at an early stage.

**Treatment.** *Prophylactic:* Breathing exercises to expand the base of the lung should be carried out during convalescence in every case of pneumonia, bronchopneumonia or empyema. Great care should be taken in operations on the nose and throat to prevent the inhalation of tissue from the operation site.

*Curative:* Cases should be diagnosed early (in the pre-bronchiectatic stage), and medical treatment instituted at once, thus avoiding the necessity of severe surgical operations. Postural drainage, as described below, should first be tried for 3 to 4 weeks. If unsuccessful, as judged by the sputum or breath remaining offensive, an attempt should be made in unilateral cases to collapse the affected lung by an artificial pneumothorax. If this can be effected the lung should be kept collapsed for 3 to 5 years, when there is every hope of a complete cure. If this

is not feasible owing to the bronchiectasis being long standing, the question of phrenic paralysis or of lobectomy or pneumonectomy must be decided. Phrenic crush or avulsion may be successful in cases of localised basal bronchiectasis. After phrenic avulsion the diaphragm on the affected side is paralysed, and rising 1 to 2 inches, produces some collapse of the base of the lung. Lobectomy or pneumonectomy may be advised for bronchiectasis confined to one lobe of the lung or to one lung but these operations are very serious. In cases of bronchiectasis due to the inhalation of a solid foreign body, bronchoscopy, with removal of the causative object, should be performed if possible. Repeated bronchoscopic drainage does not afford much hope of a cure. If the bronchiectasis is bilateral the outlook is more unfavourable, and only medical measures are available. These consist in the use of antiseptics, such as creosote, postural drainage and vaccines. Creosote may be given by mouth in capsules in doses of  $m\ 2$  to  $5\ t\ d\ s$ . It may be inhaled from a Burney Yeo mask, placing on the pad of the mask, every hour, 2 or 3 drops of a solution containing Creosot 2 parts, liq iodi mit 1 part, sp chlorof 2 parts, sp æther 1 part and phenol 2 parts. If the apparatus is available the patient may have a course of creosote baths. He is placed in a special closed chamber, the eyes are protected by goggles and the nose plugged with cotton wool. An ounce of creosote per 170 cubic feet of room space is then placed in a metal dish and vapourised over a lamp. The patient inhales the vapour for 10 to 15 minutes 2 or 3 times a week, and the cough produced results in the expectoration of a good deal of the contents of the bronchiectatic cavities. Postural drainage consists in lying, morning and evening, at first for 5 to 10 minutes with the head low down and the affected part of the lungs on a higher plane. The patient gradually becomes accustomed to postural drainage for several hours a day, and may even sleep in this position. In this way coughing is provoked and some of the pus is expectorated. A special Nelson bed may be used for this purpose (see Fig 6) or more simply a wooden frame may be made which is adjustable at different angles. A mattress is spread over this and the patient lies face downwards on it. Very satisfactory results can be obtained by postural drainage carried out for prolonged periods, the bronchi becoming dry and the patient gaining weight and losing all toxic symptoms. Breathing exercises also help to increase the movement and circulation through the lungs. Autogenous vaccines are not of great practical value. In conclusion, the importance of early diagnosis and efficient therapy cannot be overemphasised.

### Bronchial Diverticula

Bronchial pouches, resembling those described as tracheal diverticula (see p 123), are rarely seen.

### Asthma

#### (*Spasmodic Asthma*)

**Definition.** Paroxysmal attacks of dyspnoea, chiefly expiratory in nature, associated with bronchial spasm.



**Etiology.** The allergic diathesis is the most important factor. There may also be an undue irritability of the broncho-motor portion of the vagus nucleus or of the bronchial muscles themselves.

*Spasm of the bronchi* may result from : 1. Stimulation of the nucleus from psychical impulses, such as emotions, fatigue, or the sight of artificial flowers. 2. Reflex stimulation, especially from the nose, and at times from the eyes, stomach, intestines, kidneys and genitalia. 3. Bronchial stimulation from the inhalation of cold air. 4. Injected substances, such as acetyl-choline, carried by the blood to the bronchi. 5. Inhibition of the sympathetic nerve supply, as by endocrine influences.

*Edema of the bronchi* may result from : 1. Inhaled allergic substances such as pollens, room dust, book dust, orris-root powder, animal emanations, flower emanations, drugs, etc. 2. Blood-borne allergic substances such as digestive products of foods, especially of eggs, milk, fish, cheese, etc., drugs taken by mouth, bacterial products liberated from foci of infection, injected serums and skin-testing materials.

*Relaxation of the bronchi* may result from : 1. Stimulation of the sympathetic nerve supply. 2. Inhalation of substances such as stramonium and adrenaline. 3. Blood-borne stimuli such as adrenaline which has been injected or ephedrine taken by mouth (see Fig. 7, facing p. 134).

**Predisposing Causes :** 1. Heredity : Asthma often runs in families, the allergic diathesis being inherited. 2. Sex : Males predominate. 3. Age : Attacks usually start in childhood or early adult life.

**Pathology.** During the attack there may be constriction of the bronchial muscles with expiration, and, in addition, hyperæmia or œdema of the mucous membrane, with an increased output of mucus at the end of the attack. The lungs become overdistended as the air enters comparatively easily, but is expelled with difficulty. This leads to emphysema. Post-mortem no pathological changes are found in the lungs, but there may be secondary emphysema and dilatation of the heart.

**Clinical Findings.** The patient may give a history of eczema in childhood, or of a severe attack of bronchitis or bronchopneumonia which has been followed by asthma. In a typical case the attacks occur by night, the patient going to bed apparently well and waking about 2 a.m. with shortness of breath. He sits up in bed or may try to get near an open window. There is great distress.

**On Examination : During an attack.** The patient is usually sitting forward with the head resting on the hands and the elbows on the knees. The face is pale and the expression anxious or alarmed. There may be cyanosis of the lips and ears. The breathing is laboured, short and jerky, inspiration being followed by very prolonged and feeble expiration. The accessory respiratory muscles come into play. The chest. Inspection : The movements described above will be noted. Palpation : Rhonchal fremitus may be felt. Percussion : The note is hyperresonant and the cardiac and liver dulness may be diminished. Auscultation : Expiration is prolonged, rather high-pitched and wheezy, and scattered rhonchi may be heard. Vocal resonance is usually diminished. The

pulse is rapid and often weak during the attack. The blood may show an eosinophilia, up to 50%.

The attack usually stops gradually with cough and some expectoration. In the sputum Curschmann's spirals may be found, consisting of small coiled threads of mucus with some leucocytes and eosinophil cells. Octahedral Charcot Leyden crystals formed of spermin phosphate may also be present in the sputum. The attack may last for 2 hours or longer, and the patient remain wheezy for 1 or 2 days. After the attack he often falls asleep exhausted. *Between the attacks* There may be no abnormal signs found in the lungs, but in a long standing case there is usually emphysema and the chest is deformed, the shoulders being high and square and the upper dorsal spine kyphotic.

**Differential Diagnosis** Other types of paroxysmal dyspnoea must be excluded. Cardiac asthma. There is usually a lesion of the heart with or without congestive failure. The breathing tends to be less laboured, but more rapid (see p 226). Renal asthma. There is usually a history of chronic nephritis, the urine contains albumin, and relief is obtained by giving alkalis to overcome the acidosis. Bronchial asthma. The patient is a subject of chronic bronchitis and suffers from periodical attacks of asthma. Other causes of dyspnoea, such as an enlarged thymus or a loculated spontaneous pneumothorax compressing the upper half of the sound lung when the other one is extensively damaged by fibrosis, may closely simulate asthma.

**Course and Complications** The course in adults is usually progressive, the attacks becoming more frequent unless some effective means are found for checking them. In some cases an attack may last for 2 or 3 weeks, a condition designated as the *status asthmaticus*. In children asthma may cease spontaneously for no apparent reason. Complications include emphysema, bronchitis, and dilatation of the heart. Subcutaneous emphysema is a rare complication, probably due to rupture of distended marginal vesicles. Pulmonary tuberculosis may be associated with asthma. Asthmatics are especially liable to anaphylaxis, and serum should not be administered for the treatment of other diseases without preliminary desensitisation.

**Prognosis** This is good during an attack, as although the patient often looks and feels moribund death rarely occurs. Asthma, however, tends to shorten life owing to the complications, and is a very crippling disability.

**Treatment** *During the attack* At the first symptom 2 to 4 of liq adrenal hydrochlor should be injected subcutaneously. The attack can often be aborted by this means. Adrenaline can also be administered as an endobronchial application. A very fine nebulisation is required which is effected by a special spray or inhalation apparatus. With some patients a tablet of ephedrine hydrochloride gr  $\frac{1}{2}$  taken by mouth will ward off an attack. If these means fail a subcutaneous injection of 1 ml of 1 : 1000 adrenaline should be given. This contains pituitary extract and adrenaline. If no relief is obtained other remedies should be tried, such as placing a hot water bottle to the feet and drinking a cup of strong coffee, or taking liq ext grindelae m 20 in coffee every

20 minutes for 3 doses, or Eupnine Vernade (elix. caffein. ioidid. B.V.II.) m. 60 in water. An inhalation of amyl nitrite m. 5 from a capsule sometimes gives relief. Other substances used for inhalation include stramonium cigarettes or a stramonium powder consisting of stramon. fol. gr. 120, anis. fruct. gr. 60, pot. nitrat. gr. 60, which is ignited in a saucer. In very severe cases it may be necessary to give an inhalation of chloroform. For the *status asthmaticus* a syringe should be filled with 2 mls of liq. adrenal. hydrochlor. and m. 2 injected subcutaneously every 5 minutes, keeping the needle in position; the attack may be terminated in some cases in half an hour by this method. Some patients suffering from the *status asthmaticus* are not relieved even by repeated injections of adrenaline. In these cases 90% oxygen, or a mixture of 20% oxygen and 80% helium, should be given by the B.L.B. mask (see p. 143). Helium is biologically an inert gas with a low molecular weight, and mixed with oxygen in the above proportion it is about one-third as heavy as 90% oxygen. A reduction of respiratory effort of about 25% to 50% is obtained by its use. It should be given for 45 minutes followed by 90% oxygen for an hour or so. The treatment can be repeated as required for 3 or 4 days. In this way relief can be obtained for the over-distention of the lungs which occurs in severe asthma. Injection of morphine and inhalations containing cocaine are deprecated owing to their habit-producing propensity. *Between the attacks*: A thorough examination of the patient should be made. A search for a septic focus should first be carried out, with investigations of the throat, cranial sinuses and antra, teeth, faeces and urine. The lungs should be examined radiologically to determine the degree of emphysema and to exclude tuberculosis. The vital capacity should be estimated. The sputum should be examined for tubercle bacilli and for the predominating organisms, and a vaccine prepared in cases associated with bronchitis. The initial dose should be small, not more than 50,000. The dose is gradually increased at intervals of 7 days up to  $\frac{1}{2}$  to 1 million, care being taken not to provoke any reaction, which may have dire results. A fractional test meal should be performed. In about 50% of cases of asthma the acid secretion is low. If achlorhydria is present acid hydrochlor. dil. in doses of m. 30 to 60 should be given by mouth (see p. 492). The basal metabolic rate should be determined, as in some cases it is low, and the asthma is relieved by the administration of thyroideum. An investigation should be carried out as to the relation of the asthma to locality, foods, seasons, animals, flowers, plants and injections. The cutaneous protein reactions should be determined. Many cases of asthma are due to room dust or feathers, and some to face powders and cosmetics. The bedroom should be sparsely furnished, with no carpet, no covering to the chairs, and the lightest of curtains. Kapok pillows, bolster and quilt, and a rubber mattress should be used. The room should be kept scrupulously free from dust with a vacuum cleaner. In bronchial asthma, in addition to the vaccine, a course of potassium iodide and stramonium should be given, such as Pot. iod. gr. 3, tnc. stramon. m. 10, ext. glycyrrhiz. liq. m. 20, sp. chlorof. m. 5, aq. ad fl. oz. 1. Fl. oz. 1 t.d.s. This should be

taken for a month and then continued once or twice daily if it affords relief. In cases of allergy other than bacterial, it is best, if possible, to avoid the substances to which the patient is sensitive. Face and talcum powders free from orris-root are obtainable. Specific desensitisation and non-specific desensitisation by substances such as peptone and whole blood injections are not very satisfactory. An enquiry should be made as to whether or not the patient is sensitive to aspirin. If he can take aspirin without cardiac disturbance, 1 or 2 tablets at night will sometimes prevent an attack where other measures have failed. In children it has been found that pseudo-ephedrine (which is dextro-rotatory, whereas ephedrine is laevo-rotatory) gr.  $\frac{1}{2}$  b.i.d. will sometimes ward off attacks. Expiratory breathing exercises are of value in all cases of asthma, and a course, as described by the Asthma Research Council, should be carried out. No rules can be laid down as regards locality for residence, as individuals vary so much, but the majority of asthmatic patients are free from attacks at an altitude of over 4,000 feet. An asthmatic patient who is definitely emphysematous will probably be unable to tolerate such an altitude.

### Bronchial Obstruction

**Etiology.** The obstruction may result from causes within the bronchi, changes in their walls, or pressure from without. 1. *Within the bronchus*: The obstruction may be due to an inhaled foreign body, to substances entering through a tracheotomy wound, or to a gland ulcerating through the bronchial wall. 2. *In the wall of the bronchus*: Tuberculosis or syphilis may cause fibrosis, or a scar may result from a foreign body which has been removed, or from inhaled irritants. Adenoma and bronchial carcinoma are important causes. Muscular spasm is a factor causing obstruction in asthma. 3. *Outside the bronchial wall*: Pressure on the bronchi may be exerted in the mediastinum by enlarged glands, a tumour, an aneurysm, an abscess, carcinoma of the œsophagus, or by a pericardial effusion. Intrapulmonary tumours may also cause bronchial obstruction.

**Pathology.** Bronchial obstruction leads to varying degrees of collapse of the pulmonary territory. If the obstruction is due to a septic foreign body, then bronchitis, bronchopneumonia, bronchiectasis, pulmonary abscess, or gangrene may ensue. An inhaled foreign body is rather more likely to pass to the right lung than to the left, as the junction of the right bronchus with the trachea is slightly wider, and at a less acute angle than is that of the left bronchus.

**Clinical Findings.** If the patient has inhaled a foreign body, which lodges in a bronchus, he usually complains of cough and perhaps a little discomfort in the chest. A variable latent period may now occur before septic symptoms appear. Often the first symptom noted is that the breath is offensive after coughing, and then a little offensive sputum is brought up. The patient becomes more acutely ill with cough, expectoration and fever.

**On Examination:** A small area of dulness and bronchial breathing with a few fine râles may be found. The physical signs in any individual

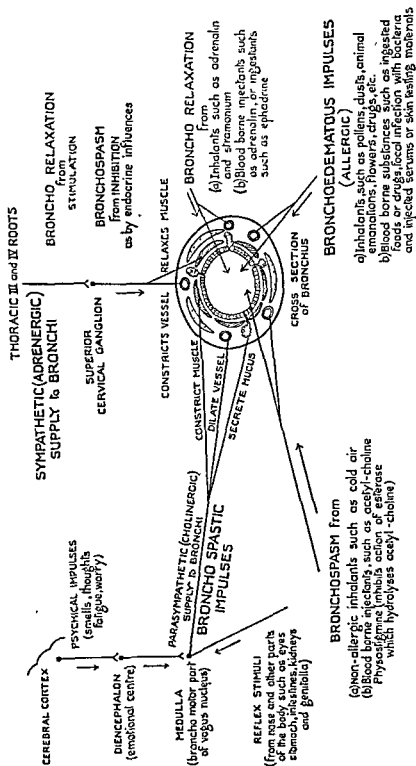


FIG. 7. DIAGRAM OF THE PATHS OF IMPULSES IN ASTHMA.



FIG. 8. RADIOGRAM AFTER AIR REPLACEMENT OF FLUID AND LIPIODOL INJECTION, SHOWING HYDROPNUMOTHORAX AND A BLOCKAGE OF THE BRONCHUS TO THE LEFT LUNG DUE TO CARCINOMA OF THE LUNG.

case vary with the degree of obstruction and the amount of sepsis. Thus a foreign body, such as an acorn, may obstruct the orifice of the main bronchus going to one lung. The whole lung will then collapse, with consequent diminution of expansion on that side of the chest, weak or absent air entry, increased voice conduction and displacement of the heart to the affected side. In other cases signs of pulmonary abscess or of bronchiectasis develop. The chest should be X-rayed and in this way a solid foreign body may be revealed. A Lipiodol injection and X-ray will indicate the site of bronchial obstruction. If this is intra-pulmonary and not due to a foreign body, bronchoscopy will probably be necessary to decide whether the bronchial obstruction is due to an intra-bronchial cause, such as a primary carcinoma of the bronchus or syphilitic stricture, or to an intra-pulmonary but extra-bronchial cause, such as a tumour of the lung.

**Differential Diagnosis.** There is usually little difficulty in diagnosis in cases of an inhaled foreign body. A history of an operation on the nose or throat, which is shortly followed by cough with fetid breath and offensive sputum, is typical of bronchial obstruction due to an inhaled septic foreign body. Bronchial obstruction due to other causes is usually diagnosed by means of the investigations detailed above.

**Course and Complications.** These vary with the cause, the complications have been mentioned above.

**Prognosis.** A foreign body usually causes little trouble if it is a hard object which can be readily removed. The outlook is serious with soft and septic foreign bodies. The prognosis is hopeless with cases due to a new growth.

**Treatment.** A hard foreign body should be removed with a bronchoscope. Obstruction due to a syphilitic stricture should be treated with a full course of iodides and mercury, such as Pot. iod. gr. 10 to 30 and liq. hydrarg. perchlor. m. 20 t.d.s. for periods of 2 to 3 months at a time, followed by a course of neoarsphenamine (see p. 570). The treatment of the other varieties should be directed to the causative factor.

### Tumours of the Bronchi

**Simple Tumours.** These include adenoma, papilloma, lipoma, myxoma, fibroma and chondroma.

**Malignant Tumours.** The growth is usually a primary carcinoma. Secondary carcinoma may also involve the bronchi. Sarcoma is rare.

### Adenoma of the Bronchus

**Pathology.** A small polypoid tumour arises, usually in mucous glands in a main bronchus, but occasionally in a tertiary bronchus. It is generally smooth, shiny and very vascular, and the greater part of the tumour is situated in the wall of the bronchus, only a small portion projecting into the lumen. It was formerly mistaken for carcinoma owing to the staining peculiarities of certain of its cells and

their irregular distribution in the connective tissue Metastases do not occur

**Clinical Findings** The patient is usually an adult under the age of 40 It is more common in women Haemoptysis of varying degree is often the earliest symptom In other cases dry pleurisy, pleural effusion or empyema may be first noted If the tumour causes bronchial obstruction there is wheezing pulmonary collapse, or bronchiectasis

**Differential Diagnosis** This includes other causes of haemoptysis, pulmonary collapse or bronchiectasis In cases complicated by pleural effusion the underlying cause is likely to be overlooked The diagnosis is established by microscopical examination of a piece of the tumour removed through a bronchoscope

**Prognosis** Cases diagnosed early can usually be treated successfully

**Treatment** There is a risk of fatal haemorrhage if removal is attempted by forceps through a bronchoscope or by diathermic coagulation Deep X ray treatment is more successful than is the insertion of radon seeds through a bronchoscope and the latter treatment may lead to bronchial stricture and pulmonary sepsis Lobectomy may be required to prevent or cure intrapulmonary suppuration or bronchiectasis

### Primary Carcinoma of the Bronchus

**Etiology** The cause is not known, but the incidence of carcinoma of the bronchus has increased during recent years *Predisposing causes* 1 Age The maximum incidence is between 40 and 55 years 2 Sex Males predominate 3 Inhalation of irritants, such as tar, asphalt, tobacco smoke, petrol fumes and radio active dust from certain mines

**Pathology** It is now generally agreed that primary carcinoma of the lung actually arises in a bronchus from an undifferentiated cell in the basal layer of the epithelium The tumour may be squamous celled, an adenocarcinoma or an undifferentiated round cell spindle cell or oat cell growth In about 75% of cases the tumour arises in a main stem bronchus and is then usually squamous celled, the remainder originate in the small peripheral bronchi, the squamous celled variety preponderating to a lesser degree Central necrosis and involvement of the pleura or thoracic wall tend to occur in the peripheral variety Tumours situated centrally may rapidly spread to the mediastinal tissues giving rise to a hilar or mediastinal type In other cases diffuse infiltration of lung tissue may occur

**Clinical Findings** The patient is usually a middle aged adult who has not previously suffered from cough In the early stages he appears in good health but now complains of a persistent dry, irritating cough Soon a little mucoid sputum appears and wheezing may be an early symptom In the peripheral type especially a few streaks of blood or a small clot may be coughed up, or pain in the chest may be complained of When the disease becomes more advanced certain clinical types can be recognised (a) *Dependent on the primary growth* 1 *Pulmonary*



The chief symptoms are cough, expectoration, pain in the chest, and dyspnoea. There is loss of weight, anorexia, sweating, and febrile attacks. In some cases in which infection has occurred the onset is acute, resembling influenza, bronchopneumonia or lung abscess. 2. *Mediastinal*: The clinical picture resembles that of mediastinal pressure or of right-sided heart failure. There may be dyspnoea, a brassy cough, an asthmatoïd wheeze, cyanosis, venous engorgement, and œdema of the legs. (b) *Dependent on the secondary deposits*. The primary lung tumour remains small and symptomless and may not be revealed by X-ray examination. 1. *Nervous*: Secondary deposits in the brain may suggest a primary cerebral tumour, hæmorrhage or abscess, encephalitis or meningitis. Pressure of secondary deposits may also cause brachial or sciatic neuritis. 2. *Ossous*: Pains in the bones or spontaneous fractures occur in about 30% of cases. 3. *Gastro-intestinal*: Metastases in glands or in the liver may result in dysphagia, jaundice, hæmatemesis or melæna. 4. *Lympho-glandular*: Enlargement of the supra-clavicular, axillary or cervical glands may simulate Hodgkin's disease.

*The Pancoast tumour* is the name applied to an apical or superior pulmonary sulcus carcinoma. The characteristic features are pain in the region of the scapula and inner side of the arm, with wasting of the small muscles of the hand. There is also homolateral cervical sympathetic paralysis (Horner's syndrome). Dulness is found at the apex of the lung and X-ray examination shows a small circumscribed apical opacity with destruction of the posterior part of the first three ribs.

*On Examination*: In the early stage no physical signs can usually be found, and in the main stem type, before any pulmonary collapse has occurred, X-ray examination of the chest is usually negative. With a peripherally situated growth X-rays may reveal a shadow due to a small area of collapsed lung. With the main stem bronchus type the physical signs which develop vary with the degree of obstruction present. With partial obstruction there is slight dulness and weak air entry over the affected part of the lung. With a ball-valve obstruction the percussion note becomes boxy from over-distention of the lung, and expiration is prolonged and wheezy. With complete obstruction the affected part collapses, with absent breath sounds and possibly displacement of the mediastinum to the affected side. In the peripheral type the signs may suggest infiltration, consolidation or abscess formation, or they may be those of dry pleurisy or of pleural effusion. In the latter case exploration may reveal a serous, hæmorrhagic or purulent effusion. With the central type the physical signs in the lungs may be very slight, over a root area posteriorly. Pressure may cause collapse, often of a lower lobe bronchus and further evidence of the mediastinal syndrome may be revealed (see p. 199). Later, the signs of infection of bronchi, or of spread of the growth in the lung, or of metastases may be found. The mass of growth may extend so that it is palpable above the clavicle, or it may ulcerate through the chest wall. The sputum: This may be scanty and tenacious, or it may be red, resembling "red currant jelly," or darker like "prune juice." Carcinoma cells have

been found in the sputum in about 60% of cases by some observers. The blood. In the later stages there is a microcytic anaemia. Leucocytosis is not the rule unless there is secondary infection. X ray examination. The shadow of the growth is only revealed in a minority of cases. There is more often a homogeneous shadow due to an atelectatic area of lung, and with infection this may become mottled or show cavity formation, the edges being hazy due to surrounding pneumonitis. The mediastinum may be displaced slightly to the affected side and the corresponding diaphragm raised. The picture may be obscured by a pleural effusion, in such instances the fluid should be aspirated and replaced by air, when the growth may be revealed by X ray examination. An X ray after intratracheal injection of Lipiodol or Neo-Hydriol (ol iodisat B P Add) may then reveal a blocked bronchus (see Fig 8, facing p 135). Bronchoscopy. This should be advised in all suspected cases of early carcinoma. In about 25% of cases, however, the growth is not visible owing to its peripheral distribution. In some cases of peripheral growth thoracotomy may be necessary to enable a specimen to be removed for microscopical examination.

**Differential Diagnosis.** This is difficult in the early stages. A persistent dry cough should not be dismissed as of no consequence, and wheezing should not necessarily be attributed to asthma. If the symptoms suggest an early lesion, bronchoscopy should not be postponed until the development of advanced signs renders radical treatment impossible. The Wassermann reaction should be determined to eliminate the possibility of a gumma. The satisfactory response to anti-syphilitic treatment in cases which give a positive reaction will help to confirm the diagnosis of a gumma. The presence of tubercle bacilli in the sputum does not negative the possibility of bronchial carcinoma, as tuberculosis and carcinoma may coexist. This also applies to the presence of a lung abscess. In cases of mediastinal obstruction it may be impossible, even with the aid of all the diagnostic tests available, to differentiate between an aneurysm and a new growth. The chief symptoms may be distal due to secondary deposits, as described above. Glycosuria may be discovered on routine examination, caused by secondary deposits in the pancreas. The irregular fever may suggest malignant endocarditis or Hodgkin's disease.

**Course and Complications.** The course is steadily progressive. Secondary deposits are liable to occur in glands, the liver, the kidneys, the suprarenals, the brain, the spinal cord, and in bones. Complications include bronchitis, bronchiectasis, lung abscess and gangrene, pleural effusions, and hæmoptysis which may be fatal.

**Prognosis.** Death usually occurs in 8 to 22 months from the onset of symptoms.

**Treatment.** In early cases, in which there are no secondary deposits, the whole lung may be removed by dissection pneumonectomy. Promising results have been obtained, the patient in some instances being well 6 years after the operation. An early tumour in the lower lobe can at times be removed by lobectomy. The use of radium, radon seeds implanted in the tumour, and deep X ray treatment offer no hope of

cure. Treatment in advanced cases is palliative, by sedatives such as diamorphin. hydrochlor. gr.  $\frac{1}{2}$  as required, for relief of cough and pain. Dyspnoea due to a pleural effusion may be relieved by aspiration and air replacement.

### Injury to the Bronchi

Rupture of a bronchus may result from severe external trauma to the chest wall, such as in a crushing accident. This is rapidly followed by surgical emphysema of the chest and neck, and death usually ensues in a short time.

## THE LUNGS

### Lobar Pneumonia

(*Croupous Pneumonia. Pleuro-pneumonia*)

**Definition.** An inflammatory consolidation of one or more lobes of the lungs.

**Etiology.** Lobar pneumonia is usually caused by the *Diplococcus pneumoniae* (pneumococcus). Thirty-two types of pneumococci have now been described. The type specificity is due to polysaccharides in the capsule of the organisms, which differ in chemical constitution in Types I, II, and III. Types I, II, and III are fairly well defined. Types I and II account for 50% to 70% of cases of pneumonia. *Predisposing causes*: 1. The presence of pneumococci in the naso-pharynx. Probably over 50% of normal individuals are carriers of pneumococci. 2. Age: Children, young adults and old people. 3. Sex: Males predominate. 4. Season: Autumn and winter. 5. A previous attack: This predisposes to subsequent attacks of lobar pneumonia. 6. Debility: Due to exposure, overwork or alcoholism.

Lobar consolidation may also be met with as the result of infection with other organisms, such as the *Bacterium typhosum* (*B. typhosus*), the streptococcus, the staphylococcus, the *Mycobacterium tuberculosis* (*B. tuberculosis*), the *Pasteurella pestis* (*B. pestis*), the *Hæmophilus pertussis* (*B. pertussis*), the *Bacterium friedländeri* (pneumobacillus) and the *Neisseria gonorrhoea* (gonococcus). The consolidation produced is a variety of secondary pneumonia.

**Pathology.** Infection probably occurs primarily by inhalation, the organisms then passing to the blood and being excreted in the urine. The pneumococci give rise to characteristic pulmonary changes. In the first stage there is active hyperæmia, the affected lobe of the lung is enlarged, but floats in water. The second stage is that of red hepatisation. The lung is solid and somewhat resembles liver. It sinks in water. The alveoli are filled with a fibrinous exudate containing red cells. The third stage is called grey hepatisation. The lung is paler, but still sinks in water, and many pus cells are present in the alveoli. The fourth stage is that of resolution, the alveolar exudate is absorbed or expectorated. It is rare for an abscess or gangrene of the lung to develop. Dry pleurisy is always found over the affected lobe. The right lung is more often involved than the left, and the lower lobes than

the upper. The inflammation probably begins at the root and spreads out into the lung.

**Incubation Period.** This is probably from 1 to 2 days

**Clinical Findings** The patient is often an adult male of rather a robust type. There may be a history of exposure to cold or wet, but rarely of contact with another patient suffering from lobar pneumonia. He is suddenly taken ill with shivering, malaise and stabbing pain in the chest. The pain may be referred to the abdomen or to the tip of the shoulder if there is diaphragmatic pleurisy. A short dry and painful cough usually rapidly develops. In children the onset is often with a rigor or with vomiting.

**On Examination** The patient looks ill, with a somewhat anxious expression, dry skin, flushed face and bright eyes. The breathing is shallow and rapid, and a grunting noise may be heard with expiration, the *alæ nasi* muscles may be seen in action. The temperature is usually high,  $102^{\circ}$  or  $104^{\circ}$  F, the pulse frequent, 110 to 120, and the respiration rapid, 30 to 40. The pulse respiration ratio may be 3:1, or even 2:1. The chest. There is diminished expansion on the affected side, the percussion note may be slightly impaired over one lobe, the air entry there is weak, and a few fine râles or pleural crepitations may be heard. X-ray examination at this stage may reveal some deep seated consolidation, which spreads outwards later. Usually on the second or third day definite signs of consolidation are found in the affected lobe. These signs are dullness, increased tactile fremitus, tubular breathing and fine "indurated" crepitations. The percussion note at the apex of the lung above the affected lobe may be *skodac*. The urine may contain a trace of albumin, with diminution or absence of chlorides. A small amount of tenacious blood stained ("rusty") sputum is usually brought up on the second or third day, and the sputum continues during the course of the illness, but gradually becomes looser and free from blood. In other cases there may be a definite hæmoptysis with bright blood. Labial herpes is often seen during the early stages of the illness. The blood pressure is generally low, and the blood count shows a leucocytosis of 20,000 to 30,000 per c mm. in "sthenic" types with a good reaction.

The temperature remains raised for 5 or 6 days, and then may fall to normal rapidly by crisis, or more gradually by lysis. At the crisis the patient's condition generally improves, there is sweating, and the toxic symptoms usually abate, but the patient remains exhausted, and collapse may occur with signs of heart failure. The physical signs of consolidation remain unchanged.

Shortly after the crisis the patient may become maniacal, but this does not usually persist for more than a day or so. As resolution occurs in the lungs there is an increase of the crepitations heard ("reduced" crepitations) and the signs of consolidation gradually disappear.

*Certain varieties of Pneumonia are described* 1. *Abortive pneumonia (maladie de Weil)* Here the onset suggests pneumonia, but no definite signs of consolidation are found in the lungs, and the temperature falls to normal in 24 to 36 hours, with rapid recovery.

2. *Apical pneumonia*: This is not uncommon in children. The physical signs in the lungs are very slight in the early stages, and the clinical picture resembles that of meningitis. 3. *Central or deep-seated pneumonia*: The symptoms resemble those of lobar pneumonia, but the signs of pulmonary consolidation are not apparent for several days. 4. *Massive pneumonia*: The bronchi are obstructed by a fibrinous exudate, and the physical signs are suggestive of a pleural effusion, although the degree of cardiac displacement is slight. 5. *Traumatic pneumonia*: Hæmorrhagic consolidation of the lung may shortly follow local trauma to the chest wall either on the ipsilateral or contralateral side. 6. *Post-operative pneumonia*: This must be carefully differentiated from massive lobar collapse (see p. 169).

**Differential Diagnosis.** The diagnosis of lobar pneumonia is often a matter of no difficulty. In the early stages it may be confused with the onset of various acute illnesses, especially influenza and enteric fever. The acute pain in the side suggests pleurisy; and this is almost invariably present in pneumonia. The subsequent course of the disease enables the diagnosis of pneumonia to be made. The initial fever and vomiting may suggest the onset of scarlet fever in children, but the diagnosis is soon established in the latter case by the appearance of the characteristic rash. Apical pneumonia may be mistaken for meningitis. The onset and early stages of acute pneumonic tuberculosis and of lobar pneumonia are very similar. In the former the temperature does not fall after a week or so, as it does in pneumonia, and tubercle bacilli are usually found in the sputum. When the pain in pneumonia is abdominal it may suggest an acute abdominal lesion, such as appendicitis. The increase in the respiration rate in pneumonia is an important diagnostic sign. Massive collapse of the lungs was formerly usually mistaken for post-operative pneumonia, but the displacement of the heart to the affected side in massive collapse and the course of the disease serve to differentiate the two. In paroxysmal tachycardia acute congestion may be found at the base of a lung suggesting early pneumonia, but a careful enquiry into the history of the illness and clinical examination establish the diagnosis. Early maniacal symptoms in lobar pneumonia may lead to an error in diagnosis. Confluent bronchopneumonia may simulate lobar pneumonia. The signs in the lungs are, however, more patchy in their distribution, the fever is more irregular, and a crisis does not occur.

**Course and Complications.** The usual course has been described above. A crisis may be expected in about 60% of cases, usually about the seventh day. Hippocrates noted that the crisis most frequently occurred on an odd-numbered day. The crisis may be as early as the third day or not until the eleventh or twelfth day. After reaching normal a small post-critical rise of temperature may occur. In some instances there is a pseudo-crisis, the temperature falling rapidly, but rising again before normal is reached. This may be due to spread of the disease (creeping pneumonia). In lysis the gradual fall of the temperature to normal may take 3 or 4 days. The following are the most important complications: Delayed resolution, pleural effusion (serous or purulent),

an empyema may develop during the acute stage of the pneumonia (syn pneumonic) or after the temperature has fallen to normal (metapneumonic) pericarditis (dry, serous or purulent), otitis media, endocarditis, meningitis, venous thrombosis, nephritis, colitis, peritonitis, jaundice, arthritis, parotitis, peripheral neuritis, abscess or gangrene of the lung. Fibrosis of the lung and bronchiectasis may occur as sequelæ.

**Prognosis** This is serious, but the mortality has been lowered from about 30% to about 8% to 11 % by the use of Sulphapyridine and Sulphathiazole. The following are unfavourable factors. The extremes of age (the mortality rate for adults under 40 years of age is one third of that for patients over the age of 40), a history of chronic alcoholism, obesity, coexisting diseases such as heart lesions, nephritis or diabetes mellitus, pregnancy, a severe degree of toxæmia, a low degree of fever and absence of response ("asthenic" type) as shown by a leucocyte count below 10 000 per c mm. A positive blood culture is always of grave import, as is also the presence of a capsular polysaccharide in the serum. Double pneumonia or creeping pneumonia is more serious than when the disease remains confined to one lobe. Unfavourable signs are a falling blood pressure with rising pulse rate, progressive weakening of the heart with dilatation, marked cyanosis, and a respiration rate of over 50 a minute. Death usually occurs from vasomotor collapse with pneumococcal bacteræmia.

**Treatment** The patient should retire to bed at the first symptom. Good nursing is essential, and a day and night nurse will be required. The patient should be isolated in an airy and well ventilated room, the temperature of which is maintained day and night between 60° to 65° F. He should lie on his back, propped up by pillows to aid the circulation through the bases of the lungs. The lumbar curve should be supported by a small pillow and the patient prevented from slipping down the bed by a pillow under the knees, attached to the bed. The clothing should not be heavy, and a pneumonia jacket of gamgee tissue with pyjamas are most suitable. The sputum should be received into a vessel containing disinfectant and covered with a lid. Tepid sponging twice a day, using water at a temperature of 90° F, is refreshing and useful if the patient is delirious. *The diet* During the acute stage the following may be given. Breakfast Milk,  $\frac{1}{2}$  pint. 11 a.m. Calves' foot jelly, Valentine's meat juice or Brand's meat essence. Lunch A cup of beef tea or meat extract. 4 p.m. Milk,  $\frac{1}{2}$  pint. 7 p.m. A cup of Benger's food, or Allenbury's Invalid diet or Horlick's malted milk. The patient should also drink plenty of fluid, such as dextrose orangeade (dextrose, oz. 4, the juice of an orange and water a pint), and barley water. Later, as the temperature falls, the diet can be augmented by thin bread and butter and a lightly boiled egg, and the milk increased. The milk may be flavoured with weak tea, as desired. The bowels should be opened daily, unless the patient is very weak, calomel gr. 8 at night followed by mag. sulph. gr. 60 to 120 next morning being given at the onset. Subsequently a morning saline aperient may be given, or Cascara Evacuant in 30 to 60 nocte, or an enema if necessary. Antiphlogistine (cataplasma kaolini B.P.) should be applied to the chest on

lint, over the affected portion of the lung and changed every 12 hours. A light gauze jacket should be worn over this. If the pain in the chest is severe it can usually be rapidly relieved by strapping the affected side of the chest in a position of full expiration, or by injecting a little air into the pleural cavity with a pneumothorax apparatus. Sleep should be secured by giving pulv. ipecac. et opii gr. 10 the first two evenings, and later by the use of paraldehyde m. 60 to 120 in m. 30 capsules. In some cases of obstinate insomnia brandy fl. oz. 1 in hot water at night acts like a charm. If the patient is an alcoholic, whisky or brandy fl. oz.  $\frac{1}{2}$  should be given four- to six-hourly. For the irritating cough a sedative linctus may be prescribed, such as Tnc. opii camph., syr. pruni serotin. and glycerin., āā m. 20; m. 60 occasionally. Later, as expectoration loosens, a stimulating mixture should be given such as Ammon. carb. gr. 5, pot. iod. gr. 3, tnc. scillæ m. 20, sp. chlorof. m. 7, infus. senegæ rec. ad fl. oz. 1. Fl. oz. 1 t.d.s. Larger doses of ammon. carb., such as gr. 15, given twice a day in a glass of milk, are also very valuable. If there is cyanosis oxygen should be given continuously through a small nasal catheter (gauge 4 English). This will permit a flow of 4 or 5 litres a minute. A double nasal catheter held in position by the Tudor Edwards spectacle frame, using ordinary cycle valve tubing (gauge 10 French) with three additional small holes cut in the last half inch of the end of the tube, will permit a flow up to 10 litres a minute. The nose and nasopharynx should be sprayed with 1 in 500 Percaine in liquid paraffin before the tubes are introduced and the tubes should be lubricated with liquid paraffin. The tubes should be passed a distance of about 3 inches to the nasopharynx. The oxygen must be moistened by bubbling through 6 inches of warm water. Large cylinders of oxygen should be used, fitted with a pressure-reducing regulator and a water or bobbin flowmeter. With the B.L.B. (Boothby, Lovelace and Bulbulian) mask the alveolar concentration of oxygen can be varied from 45% to 90%. Such high concentrations should not be used continuously for longer than 48 hours, for fear of damage to the pulmonary alveoli. There are two types of mask, nasal and oro-nasal, the latter being used by mouth breathers. An oxygen tent is of great value for babies. Signs of cardiac dilatation or weakening of the pulse should be treated by the administration of such drugs as digitalin gr. 1/100 subcutaneously six-hourly, Cardiazol (leptazolium B.P. Add.) 1 mil., or Coramine (nikethamidum B.P. Add.) 1.5 mil. subcutaneously four-hourly, or liq. adrenal. hydrochlor. m. 5 subcutaneously six-hourly. The most effective respiratory stimulants are strychnin. hydrochlor. gr. 1/60 hypodermically six-hourly, lobelin. hydrochlor. mg. 10 in 1 mil. hypodermically repeated three times in 5 hours, if necessary, or carbon dioxide 5% in oxygen, by inhalation. Flatulent abdominal distention must be treated early, as it embarrasses both heart and lungs. For this purpose a rectal tube should be passed, a turpentine enema given containing turpentine fl. oz. 1 in 15 fl. oz. of starch mucilage, or turpentine stupes applied to the abdomen, prepared by sprinkling m. 60 of turpentine over a hot fomentation. If these measures fail, a hypodermic injection should be given of 1 mil. of

Pituitrin (ext pit liq B P), and albumin water may be substituted for milk for a day or so. For delirium and restlessness an ice compress should be applied to the head and a rectal injection given of pot brom gr 120 in water fl oz 2. The patient must be kept lying quietly for at least 2 days after the crisis, and if sweating is very exhausting a subcutaneous injection of atropin sulph gr 1/100 can be given. Severe collapse should be treated by cardiac stimulants as detailed above, and by brandy fl oz  $\frac{1}{2}$  six hourly.

The serum treatment, described in previous editions, has now been almost universally replaced by the use of Sulphapyridine, Sulphathiazole and Sulphadiazine. Sulphapyridine (M & B 693) is given in the following doses for an adult:  $\frac{1}{2}$  tablets (each 0.5 G) repeated in 4 hours, then 2 tablets every 4 hours for 2½ days. The dose is now reduced to 1 tablet four hourly for 2½ hours and then 1 tablet eight hourly for 36 hours making a total of 2.3 G in 5 days. For children gr 1 to 1½ (0.06 to 0.1 G) per lb body weight should be given every 2½ hours. The dosage up to the age of 5 years is as follows: 1 to 3 months,  $\frac{1}{4}$  tablet four hourly; 3 to 12 months,  $\frac{1}{2}$  tablet four hourly; 2 years,  $\frac{3}{4}$  tablet six hourly; 3 years, 1 tablet six hourly, and 5 years 1 tablet four hourly. The duration of the average course is 5 to 7 days. The tablets are crushed and suspended in water or milk. It is not now considered that sulphur containing substances such as eggs, milk, and magnesium and sodium sulphate should be avoided during the treatment. The fluid intake should be sufficient to prevent the urine becoming concentrated or hematuria may result from irritation caused by excretion of crystals of the acetylated compounds of the drug. Toxic manifestations include headache, nausea, vomiting, methemoglobinemia, cyanosis and hematuria. Drug rashes, drug fever and granulocytopenia may occur if the patient is unduly susceptible or if the treatment is continued too long. If the patient vomits, the drug may be injected. A soluble sodium solution (M & B 693 Soluble) is available containing 1 G in 3 mls. This should be injected intravenously diluted to 10 mls with normal saline. The injections are given four hourly. If the treatment is efficacious the temperature usually falls to normal by lysis in 24 to 36 hours but the resolution of the pneumonic consolidation is not accelerated.

Sulphathiazole (M & B 760), given in similar doses, appears as efficacious as Sulphapyridine in the treatment of pneumococcal pneumonia but it does not cause such a rapid fall in temperature. A satisfactory method is to give Sulphapyridine for the first two days and to complete the course with Sulphathiazole. Sulphadiazine, the pyrimidine analogue of Sulphapyridine, is also very efficacious in the treatment of pneumococcal, staphylococcal and streptococcal pneumonia. It is given in similar doses and appears to be less toxic than the other two sulphonamides.

During convalescence breathing exercises must be carried out to expand the lungs. These should be inspiratory in nature, and not expiratory as in blowing through Wolff's bottles. By this means the risk of subsequent bronchiectasis is much diminished. Resolution



should be checked by X-ray examination. In cases of delayed resolution an autogenous vaccine should be given, beginning with small doses such as one million, and gradually increasing the vaccine at intervals of 5 to 7 days. Later a tonic should be given such as syrup ferri phosph. co. m. 60 t.d.s. p.c.

### Bronchopneumonia

(*Lobular pneumonia. Catarrhal pneumonia. Capillary bronchitis*)

**Definition.** Inflammatory consolidation of one or more lobules of the lungs, with acute inflammation of the terminal bronchioles.

**Etiology.** Bronchopneumonia may be primary or secondary.

**Primary Bronchopneumonia:** This may be due to infection with such organisms as the *Bacterium freidlanderi* (pneumobacillus), the *Diplococcus pneumoniae* (pneumococcus), the streptococcus, the staphylococcus, or the *Mycobacterium tuberculosis* (*B. tuberculosis*). **Secondary Bronchopneumonia:** This may occur as a complication of measles, whooping-cough, gastro-enteritis, influenza, diphtheria, scarlet fever, enteric fever, small-pox, etc. The aspiration, deglutition or septic variety results from inhalation of septic material. This may be caused by operations on the naso-pharynx, or occur in cases of cerebral tumour or bulbar paralysis, or in coma due to uræmia or cerebral hæmorrhage. Septic material may be coughed from the diseased to the healthy lung, as in bronchiectasis. The vomit may be inhaled, as in carcinoma of the œsophagus, or in a gastro-enterostomy operation on a dilated stomach which has not been emptied by a stomach tube. Lipoid pneumonia (oil aspiration pneumonia) has been described in debilitated infants. It is due to inhalation of droplets of mineral or cod-liver oil which has been given to improve nutrition. In adults it usually results from the persistent use of nasal oils. It then often assumes a chronic form suggesting chronic bronchitis or fibrosis of the lungs. Oil droplets may be found in the sputum. The inhalation of mustard gas may also lead to the secondary development of septic bronchopneumonia.

**Pathology.** **Primary Bronchopneumonia:** There is an acute inflammation of the terminal bronchioles of one or more lobules, and the distal portion of the lung is filled with an exudate. This is rich in white cells and contains relatively little fibrin. Both lungs are usually affected, and the consolidated lobules may be confluent. **Secondary Bronchopneumonia:** Similar changes occur, but in septic cases there may be more definite suppuration, leading to abscess of the lung, or more rarely to gangrene or bronchiectasis.

**Clinical Findings.** **Primary Bronchopneumonia:** The patient is usually an infant or young child. The onset is comparatively sudden, with the symptoms of a feverish cold.

**On Examination:** **Inspection:** The child looks flushed or cyanosed, restless and ill. The respirations are rapid, expiration grunting and a dry cough may be heard. The *alæ nasi* muscles are seen to be in action, and there may be recession of the intercostal spaces and retraction of the xiphisternum with inspiration. **Palpation:** Rhonchal fremitus may be felt, if there is an associated large-tube bronchitis. **Percussion:** Small

areas of impaired resonance may be detected, especially in the lower lobes behind. Auscultation. The air entry is often weak over the lower lobes and harsh over the upper lobes. Small patches of bronchial breathing may be heard, with bronchophony and a few fine crepitations. Often, however, no sign of consolidation can be detected, but rales may be heard over the lower lobes. In confluent bronchopneumonia larger areas of consolidation are present. The heart sounds are rapid, and the pulmonary second sound is usually accentuated. The temperature rises rapidly to  $103^{\circ}$  or  $105^{\circ}$  F, and then becomes remittent in type, falling by crisis or lysis in about 7 to 10 days. A further rise in temperature may indicate a fresh spread of the infection. The respirations may rise to 40 or 60 or even higher in a severe case, and the pulse rate to 120 or more. Infants have no sputum, any pulmonary secretion which is brought up on coughing being either swallowed or vomited. In severe cases the child is very drowsy, being overwhelmed with the toxæmia.

*Secondary Bronchopneumonia.* The onset is more gradual with preliminary bronchitis occurring during the course of some other illness such as measles. The physical signs of localised areas of consolidation are usually easier to detect.

*Differential Diagnosis.* There is usually little difficulty in the diagnosis, but the extreme dyspnoea may suggest laryngeal diphtheria. There is, however, no obstruction to the airway through the larynx. In some cases the cerebral symptoms may suggest meningitis. Tuberculous bronchopneumonia is not often diagnosed in a child before death although the course tends to be more prolonged and tubercle bacilli may be found in the sputum from swallowed sputum.

*Course and Complications.* In favourable cases the temperature falls in about a week and the child makes a rapid recovery. In severe cases the child may be quickly overcome by toxæmia, or the course may be prolonged by spread of the disease. There are usually no complications in primary bronchopneumonia, but in the secondary variety lung abscess, bronchiectasis or empyema may ensue.

*Prognosis.* Primary bronchopneumonia proves very fatal to young infants. The prognosis of primary bronchopneumonia in children is not, however, so serious. Unfavourable signs are dilatation of the heart, increasing cyanosis, dyspnoea and drowsiness. Death usually occurs from respiratory failure. Secondary bronchopneumonia, when complicating such diseases as measles, is also very deadly. Aspiration bronchopneumonia often causes death.

*Treatment.* The treatment of an infant aged 1 year, suffering from primary bronchopneumonia will be described. The infant must be in bed, but should be taken in the arms from time to time, to change its position. The shoulders and head should be slightly raised. The temperature of the room should be kept at  $65^{\circ}$  F day and night, and the air moistened with a steam kettle, especially in cases of obstructive dyspnoea. The chest should be lightly rubbed with camphorated oil (lin. camphoræ B.P.) and covered with a gamgee jacket and a nightdress opening down the front. If the temperature rises over  $105^{\circ}$  F, or there is much restlessness, the infant should be tepid sponged.

with water at 85° to 92° F. The diet consists of 6 to 8 oz. of equal parts of milk and water, containing 2 teaspoonfuls of lactose, every 3 hours. This is given by a spoon or by bottle. If there is curd indigestion, the feeds are citrated, using sod. citrat. gr. 2 to each ounce of undiluted milk. Drinks of half strength normal saline (0.42% sod. chlorid.) are given between feeds, and if the infant is dehydrated a continuous subcutaneous drip of normal saline is also advisable. The bowels should be opened every 2 or 3 days by syrup. flicorum m. 60 nocte, or a glycerin suppository may be used. Sulphapyridine (M. & B. 693) should be given in the doses described on p. 144. Expectorants are of little value unless given in emetic doses, when they are dangerous and are best avoided. Respiratory stimulants include strychnin, hydrochlor. gr. 1/200 injected subcutaneously and repeated four-hourly until slight muscular twitching is seen, or lobelin, hydrochlor. mg. 3 in 1 mil. subcutaneously, which may be repeated in 2 hours. For circulatory failure Coramine (nikethamidum. B.P. Add.) 0.5 mil. should be injected subcutaneously every 4 hours. For restlessness and insomnia brandy m. 20 in  $\frac{1}{2}$  fl. oz. of water may be given and repeated in 4 hours if necessary. Cyanosis should be relieved by the use of an oxygen tent.

### Pneumonitis

**Definition.** A localised area of inflammatory exudation in the lungs.

**Etiology.** Various types occur, such as the influenzal, the simple pneumonitis in childhood, the chronic pneumonitis of infancy and childhood, and the chronic pneumonitis of adults. Pneumonitis may occur as an early stage in the formation of lung abscess, especially in the aspiration variety, or it may develop around a bronchiectatic cavity or lung abscess, a bronchial carcinoma or adenoma, or it may be associated with multiple small septic emboli, as in thrombo-phlebitis migrans, or with areas of pulmonary collapse. Various catarrhal organisms are responsible for the condition and a virus pneumonitis is described in infants and adults.

**Pathology.** There is probably an exudation into the alveoli of a localised area of the lungs, with cellular infiltration of the terminal bronchioles and alveolar walls. Cytoplasmic inclusion bodies are found in the bronchial epithelial cells.

**Clinical Findings.** In the acute pneumonitis of childhood the patient suffers from lassitude and anorexia, with cough and perhaps a little expectoration. There is no history of a previous acute illness. *On Examination:* The percussion note may be slightly impaired over a portion of the chest, with weak air entry and fine or medium râles. An X-ray film shows a mottled area in the chest resembling the appearances produced by an unresolved pneumonia. The condition usually clears up in a week or two, and no special treatment is required. Cough, dyspnoea and cyanosis are the predominating features of primary epidemic virus pneumonitis occurring in infants. The treatment is as for bronchopneumonia.

## The Chronic Pneumonias

*(Chronic Focal or Disseminated Pneumonia)*

Under this term are included a number of chronic inflammatory conditions of the lungs. It embraces what are generally described as fibrosis of the lungs, chronic interstitial pneumonia, and certain circumscribed suppurative or non-suppurative pulmonary inflammations. The diagnosis is chiefly dependent on the X ray findings and the exclusion of such conditions as pulmonary tuberculosis, new growth, lung abscess, pneumonitis, bronchiectasis, etc.

## Pulmonary Tuberculosis

*(Consumption. Phthisis)*

**Definition.** Tuberculous infection of the lungs, bronchi, bronchial glands or pleuræ.

**Etiology.** Pulmonary tuberculosis is caused by the *Mycobacterium tuberculosis* (*B. tuberculosis*). Both human and bovine types affect

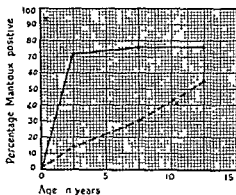


FIG 9 MANTOUX SKIN TESTS. DOW AND LLOYD 5 years is five times greater in children who are contacts  
WINGFIELD *Brit Med. Journ The Practitioner*

man. The organisms may infect the body from dust or dried sputum, by droplet infection, or by contaminated articles of food, such as milk, cream, butter, cheese or meat. Contact with "open" or sputum positive cases is probably the most important source of infection. The incidence of tuberculous infection can be determined by the Mantoux test (see p. 154), by which it has been shown that infection from birth to 5 years is five times greater in children who are contacts with sputum positive cases, than in those who are non-contacts. Further between the ages of 0 to 15 years contacts are infected twice as often as non-contacts. This is illustrated in the chart (see Fig. 9). Although only a small proportion of these infected children subsequently develop pulmonary tuberculosis, they are much more prone to do so than are those who give a negative reaction. Close contact with "open" cases of pulmonary tuberculosis, as in households or at work, is a very important cause of active tuberculosis in adults. Conjugal infection is a very real danger which has been underestimated in the past. **Predisposing causes.** 1. Heredity. The disease undoubtedly occurs in families, and in some rare instances infants may be born with tuberculosis. 2. Race: Virgin races are very susceptible, i.e., those which have not previously suffered from the disease, and which, as the result of civilisation, are brought in contact with it. 3. Sex: There is a slight predominance of males. 4. Age: Pulmonary tuberculosis is

rare below the age of 7, although glandular tuberculosis is common in children. The maximum age incidence of pulmonary tuberculosis is between 15 and 45 years. 5. Climate: Pulmonary tuberculosis is prevalent in districts exposed to rain-bearing winds. 6. Sanitation: Overcrowding and absence of sunlight and fresh air are potent predisposing factors. 7. Occupation: Inhalation of dust, especially silica particles. Tin miners are prone to the disease, whereas coal miners are relatively immune. 8. General health: Lack of food, overwork, chronic alcoholism, diabetes mellitus and measles predispose to tuberculosis.

**Pathology.** The tubercle bacilli may reach the lungs by three routes: By the respiratory tract, by the blood stream and by lymphatics. In childhood, infection is chiefly from the alimentary tract, due to infected milk. The organisms are of the bovine type, and pass by lymphatics to the bronchial glands and thence to the lungs. The bacilli may also travel from the tonsils to the cervical glands and thus to the lungs. In some cases a primary focus (Ghon) may be demonstrated radiologically in the lung parenchyma. In adult life there are two theories as regards infection. Pulmonary tuberculosis may be due to a reactivation of a childhood infection in the lungs or elsewhere, or it may be caused by a recent infection by inhalation. The second is probably the more usual occurrence, as shown by the evidence of contact infection, and the laboratory investigations which demonstrate that in the vast majority of cases the organisms are of the human type, whereas in the glandular infections of childhood the tubercle bacilli are overwhelmingly of the bovine variety. Whereas newborn infants are almost invariably free from tuberculous infection, in over 90% of autopsies on adults a tuberculous lesion is found. This is usually an old "obsolete" focus. The initial lesion is in the subpleural layers of the lung parenchyma. Tubercles form which may coalesce and produce areas of consolidation, they may then become caseous with cavity formation, or be arrested and heal with fibrosis and calcification. The mediastinal lymph glands are next involved. The lung lesion often heals while the glandular lesion smoulders. Spread may occur directly by the blood or lymph stream, or bronchogenic spread may occur from pulmonary cavities. X-ray examinations throw doubt on the traditional view that the initial lesion is at the apex of the lung. It is more often situated in the sub-clavicular region (Assman's early infiltration) or near the root of the lung. In children the primary focus, described by Ghon, may be seen in the lower lobe or elsewhere, and the associated root glands are enlarged. Lower lobe tuberculosis may also occur in adults and is not so uncommon as was at one time believed. Miliary tuberculosis of the lungs may result from a caseous bronchial gland rupturing into a blood vessel. In addition to the local lesions produced in the lungs, the tubercle bacilli liberate exotoxins, which circulate in the blood and give rise to profound constitutional disturbances.

**Clinical Findings.** Pulmonary tuberculosis may have an insidious or acute onset. The former is the more common. *The insidious onset:* The early symptoms are very numerous; amongst the most important are the following: Cough, which persists for several months, usually

with some expectoration. Lassitude and loss of weight. Palpitations or dyspnoea on exertion. Nervous debility which may simulate neurasthenia. Amenorrhœa is not infrequent. In other cases hoarseness is the earliest symptom. Some patients complain of periodical shivering or sweating. The symptoms may date back to what the patient designates an attack of influenza, or to a cold which has never properly disappeared. In children an attack of measles or whooping-cough may be followed by persistent ill health leading up to the diagnosis of tuberculosis. *The acute onset:* This may also be of several varieties. *Hæmoptysis:* The patient is apparently in good health and suddenly coughs up blood, in varying amounts. *Spontaneous pneumothorax:* Here also the patient is apparently well, when the pneumothorax suddenly occurs with severe pain and dyspnoea. *Pleurisy.* This may be acute dry pleurisy or pleurisy with effusion. *Acute influenzal type.* *Acute pneumonic or bronchopneumonic tuberculosis* ("galloping" consumption). The onset closely resembles that of lobar or bronchopneumonia. *Acute benign type.* *Acute miliary tuberculosis:* The patient is suddenly taken ill with a high fever, malaise and dyspnoea. This is more common in children than in adults.

*On Examination* 1. *Chronic pulmonary tuberculosis:* The early stages (a deposit or infiltration). There is no characteristic appearance, and the patient is not usually wasted, although the weight is often below his highest known weight. *Examination of the chest.* *Inspection.* There may be drooping of one shoulder, with slight flattening and diminished expansion of the upper part of the chest on the affected side. *Palpation:* Tactile fremitus is slightly increased over this area. *Percussion.* The note is slightly impaired at the upper part of the chest, and Krönig's area of apical resonance above the clavicle may be diminished. *Auscultation.* The breath sounds over the affected area of lung may be weak, expiration may be prolonged, or the air entry jerky. The breath sounds in other cases are broncho-vesicular or vesiculo bronchial. At the back the breath sounds in the supraspinous region may be coarse and rough (Grancher's "granular" breathing). The air entry may be weaker at the base on the affected side. Often there are no adventitious sounds, or an occasional râle may be heard after cough. There is usually slight bronchophony and faint pectoriloquy. The special signs and symptoms indicative of activity are described below (see p 153).

*Consolidation of the Lung.* The signs in the chest are more marked. The note is dull, tactile fremitus is increased, the breath sounds are bronchial, usually some coarse râles are heard, and there is bronchophony and pectoriloquy.

*Caseation of the Lung.* There is dulness with increased tactile fremitus, the breath sounds are hollow and bronchial, and after cough, showers of crackling and bubbling râles are heard. There is also bronchophony and pectoriloquy.

*Excavation of the Lung.* There is flattening of the chest wall over the cavity; the note is hyperresonant over the cavity if it contains air, and if it is in communication with a bronchus a "cracked pot" sound may be heard on percussion (*bruit de pot fêlé*), or the note may alter in

pitch as the percussion is performed with the mouth open and closed (Wintrich's sign). The breath sounds are amphoric or cavernous, and coarse or metallic râles are heard. There is intense bronchophony and pectoriloquy, and immediately after cough a hissing sound may be heard (post-tussive suction). When a large cavity contains air and fluid a coin sound and ægophony may be present. If the cavity is filled with fluid the physical signs are dullness, weak breath sounds and diminished voice conduction.

*Fibrosis of the Lung:* There is flattening of the chest wall, with diminished tactile fremitus, dullness, weak air entry and some coarse leathery or sticky râles. Vocal resonance is diminished. If the fibrosis affects the left upper lobe, pulsation may be seen in the second and third left spaces close to the sternum, caused by retraction of the lung and uncovering of the heart. The trachea may be displaced to one or other side, and this may occur without any cardiac displacement, if the fibrosis affects the upper part of the mediastinum only.

Various lesions are often combined in chronic pulmonary tuberculosis, such as caseation or consolidation with fibrosis, which is met with in chronic fibrocaceous or ulcerative tuberculosis.

2. *Chronic miliary tuberculosis:* The patient is usually between the ages of 11 and 30 years and may complain of cough, dyspnoea, pain in the chest, expectoration, and at times of hæmoptysis. The evening temperature is usually a little raised. The signs in the lungs are very slight, a few scattered râles being heard at several points. The spleen may be palpable, and glands, bones or joints may show tuberculous lesions. Diagnosis can be established in many cases by finding tubercle bacilli in the sputum, stomach washings, or in a pleural effusion if it is present. X-ray examination shows small, rather soft, nodular shadows, scattered throughout the lung fields. These were formerly diagnosed radiologically as being due to acute miliary tuberculosis. The patient, however, is often ambulant and not very ill, the course of the disease may be prolonged for a year or more, and arrest may occur. The differential diagnosis includes carcinomatosis, pneumokoniosis, sarcoidosis and periarteritis nodosa.

3. *Hilum tuberculosis:* This is usually a variety of chronic pulmonary tuberculosis in which the lesion begins near the lung root and spreads outwards in what is known as a "hilar flare." Some authorities are sceptical of its existence, but X-ray evidence indicates the probability of its occurrence in certain cases.

4. *Epituberculosis:* The lesion here is considered to be due to a tuberculous root gland causing a triangular area of collapse in the upper zone of a lung, owing to pressure on a bronchus. The alternative view is that it is a benign and retrogressive type of tuberculous pneumonia resulting from the erosion into a bronchus of a caseous gland containing few viable tubercle bacilli. The patient is usually a child or adolescent, who complains of cough. Signs of consolidation may be found, usually in the right upper lobe, which take weeks or months to disappear.

*General Examination:* The fingers are often clubbed, in the early

stages the nails being curved like a parrot's beak or puffin's bill. Drumstick clubbing is more often associated with bronchiectasis. The cheeks, ears and lips may show slight degrees of cyanosis. The temperature is usually raised if there is activity present, as described later, and the pulse is also quickened. The sputum. This possesses no characteristic appearance. It may be thin and frothy, or thick and purulent. In advanced cases it is often green and nummular, floating on water in flat circular masses about the size of a shilling. Blood may be present in streaks or clots, or the sputum may be uniformly coloured pink. An examination for tubercle bacilli should be made on several occasions in every doubtful case, but negative results do not exclude a diagnosis of tuberculosis. The sputum may also be cultivated for tubercle bacilli. In the case of children or patients with no sputum a few droplets of mucus may be coughed up and collected upon a laryngeal mirror held at the back of the throat. At times tubercle bacilli may thus be found. The blood. There are again no characteristic changes. The Bonssdorff count consists in enumerating the lobes of the nuclei of 100 polymorphonuclear cells. The normal figure is taken as 274. Lower readings are said to occur in active cases. The rate of sedimentation of the red cells in citrated blood is an indication of activity in tuberculosis, and a guide as to progress, but it is not a diagnostic test of tuberculosis. The complement fixation test is not reliable as a diagnostic guide. Tuberculin tests are only indicative of a tuberculous infection at some time in the life of the patient and not of present activity.

*X ray Examination.* This should form part of the routine examination of every suspected case of pulmonary tuberculosis, although tuberculosis cannot be diagnosed solely on a radiogram. Deep seated cavitation can best be revealed by tomography.

*5 Acute influenzal type.* Attention has been drawn to the acute form of onset resembling influenza, in which the patient is not nearly so ill as in the bronchopneumonic variety.

*On Examination.* Some râles are heard below the clavicle in the second and third spaces, and the X ray films show infiltration usually in the upper zone below the clavicle, less often in the middle or lower zone.

*6 Acute pneumonic tuberculosis.* The mode of onset resembles that of lobar or lobular pneumonia, but in some cases it is not quite so sudden. In children acute tuberculosis often follows an attack of measles or whooping-cough, whereas in adults there has usually been a pulmonary tuberculous focus for some time, as shown by fibrosis with mediastinal displacement.

*On Examination.* Signs of consolidation are generally found in the lower lobe of one lung. The temperature is raised to 101° or 103° F, and remains continuously so for a week or ten days. A crisis does not occur but the temperature begins to swing more, becoming remittent in type, and the signs of toxæmia are more evident. The cyanosis of a mauve tint is very characteristic, with tachycardia and dyspnoea. The physical signs become more those of caseation than of consolidation. The diagnosis is established by finding the tubercle bacilli in the sputum.



7. *Acute benign tuberculosis*: The patient is usually a young adult who is taken ill with what he regards as a feverish cold. There is cough and a little sputum for a few days and the sputum may be blood-stained. The temperature settles in about a week and the patient feels well. X-ray examination reveals shadows suggesting a lobar, bronchopneumonic or miliary process, and annular shadows may also be seen. The sputum often contains tubercle bacilli. The patient is not toxic, and in 6 to 8 weeks the ill-omened shadows disappear from the radiogram. The patient should be kept in bed until this occurs.

8. *Acute miliary tuberculosis*: In addition to the symptoms mentioned on p. 150, examination of the chest may reveal remarkably few abnormal signs, or there may be râles scattered through the lungs. Drenching night sweats are often a marked symptom, but there are usually no rigors. The temperature is of the remittent or intermittent type, and it may be inverted, the morning temperature being higher than the evening. X-ray examination shows changes closely resembling those found in chronic miliary tuberculosis (see p. 151) and both lungs may present a "snow-storm" appearance. The patient rapidly becomes more ill, the spleen may be palpable, and death usually occurs from profound toxæmia in 2 weeks to 3 months.

*The following are the signs indicative of active disease.* <sup>10</sup> The temperature: A rise of mouth temperature, to 99° F. or over, usually in the afternoon or evening. This may occur when the patient is at rest, or only after exercise. In some cases activity may be present when the temperature does not rise above normal, a subnormal swing being found. The pulse: Persistent rapidity at rest is usually an indication of activity. The sputum: When tubercle bacilli are present in the sputum the disease is usually, although not invariably, active. Other indications of active disease are an increased rate of sedimentation of the red cells, loss of weight, increase of cough and expectoration, night sweats, and lassitude. Cyanosis, if due to toxæmia, is also an indication of activity, but it may occur in arrested disease if there is much pulmonary damage.

*Differential Diagnosis.* The chief difficulty arises in the diagnosis of the early case. Great attention should be paid to the various symptoms detailed on p. 149. A temperature record should be obtained with the patient in bed, and, if he is afebrile, the effect on the temperature of the patient getting up and exercising should be determined. The lungs must be X-rayed, and special knowledge is required for the correct interpretation of the radiograms. It must be realised that a diagnosis of tuberculosis cannot be made on radiographic findings alone. The X-rays may reveal a shadow due to infiltration of the lung parenchyma, but this infiltration may be due to causes other than tuberculosis. In *sarcoidosis* (Desnier-Boeck-Schaumann disease) a diffuse mottling of the lungs fields may be seen on X-ray examination, resembling miliary tuberculosis. The root gland shadows may be much increased. In addition there may be enlargement of the spleen, lymph glands, parotid and lachrymal glands, iridocyclitis and infiltrations of the skin and phalanges of the hands and feet. Biopsy of a superficial lymph gland

shows the characteristic lesion, the "hard tubercle" Giant cells are frequently present containing peculiar cell inclusions The plasma globulin is increased The cause of the disease is unknown, but the pathological findings are those of a benign lymphogranulomatosis The sputum should be examined repeatedly for tubercle bacilli, and if not found the stomach washings and faeces should also be tested. Other causes of ill health which especially require exclusion are tachycardia and loss of weight due to hyperthyroidism and pyrexia due to septic foci elsewhere In cases of doubt the Mantoux intradermal tuberculin test can be applied A positive reaction only implies that the patient has been infected with tuberculosis at some period of his life, not that he has active disease now A negative reaction, on the other hand, is strong evidence against the diagnosis of active pulmonary tuberculosis Purified protein derivative (P P D) is now used in the test instead of old tuberculin (O T), as it is of constant potency It is put up in two strengths, the weaker being equivalent to 1/25,000 to 1/50 000 O T, the other being 250 times stronger 0.1 ml. of the solution is injected intradermally and the skin reaction read at 48 hours A positive reaction is shown by an area of erythema and induration measuring at least 5 mm in diameter When there are definite signs in the lungs, such conditions as bronchitis and emphysema, a new growth, actinomycosis pulmonary fibrosis due to other causes, such as syphilis or pneumoconiosis, must all be differentiated from tuberculosis The difficulty is increased by the fact that pulmonary tuberculosis may coexist with any of these conditions

**Course and Complications** *Acute caseous tuberculosis* This usually rapidly extends and proves fatal in 2 to 3 months, arrest, however, does occur in a certain proportion of cases *The acute benign type* As described above, recovery is to be expected *Acute miliary tuberculosis* This is generally fatal in 2 weeks to 3 months *Chronic pulmonary tuberculosis* A very variable course must be expected depending largely on the resistance of the patient Thus an early lesion may be almost immediately arrested by fibrosis or calcification, or it may gradually extend Periods of activity may alternate with times of comparative arrest The clinical course of the disease at any moment may be clearly illustrated by Inman's classification of the stages of the disease Stage I, the patient is febrile in bed Stage II, he is afebrile in bed, but febrile when ambulant Stage III, he is afebrile when in bed, and afebrile when ambulant Stage IV, the patient is afebrile also when working Cases are also classified as "open" or "closed," the former having tubercle bacilli in the sputum, or as A and B cases, in the former, tubercle bacilli have never been found in the sputum, either at the time of classification or at some previous date in the latter tubercle bacilli have been found in the sputum Classifications based on the pathological changes in the lungs and on their extent are not so valuable, unless combined in some way such as in Philip's method, so that an indication of activity is given

The following complications are of great importance Laryngitis catarrhal or tuberculous Pleurisy, dry or with effusion Pneumo-

thorax. Intestinal tuberculosis: In about 85% of cases of pulmonary tuberculosis with excavation, intestinal tuberculosis occurs. The lesion is usually in the ileo-cæcal region. X-ray examination usually shows that the barium does not fill the cæcum, when the ileum, ascending and transverse colons are filled. Fistula-in-ano. Meningitis. Bronchitis and emphysema. Spontaneous subcutaneous emphysema rarely occurs. Asthma. Bronchiectasis. Tuberculosis of the epiglottis, tonsils, trachea, pharynx, tongue or nose. Tuberculosis of the epididymis, prostate, bladder, peritoneum or kidneys. Venous thrombosis. Peripheral neuritis. Amyloid degeneration. Rib abscess. Myocarditis and pericarditis.

**Prognosis.** This is very grave in all forms of acute tuberculosis. In chronic cases many factors have to be taken into consideration, but in any case the average length of life after diagnosis is only about six years. The most definite prognostic guide is afforded by the presence or absence of tubercle bacilli in the sputum. The after history of patients who have left sanatoria shows that only 36% of those with tubercle bacilli in the sputum on discharge are alive  $5\frac{1}{2}$  years later. In women the greatest number of deaths occurs between the ages of 15 and 30 years, and in men between 20 and 55 years. Complications increase the gravity of the disease, especially diabetes mellitus, tuberculous laryngitis and enteritis. Meningitis is a fatal complication. Freedom from financial worry enables life to be passed under the most favourable conditions, and thus improves the prognosis. In all cases which have already received efficient treatment, the response obtained will afford a valuable guide as to prognosis. *Further the prognosis does not depend entirely upon the activity or extent of the disease, but the type of disease present is of the greatest importance.* Thus the exudative type of disease bears a worse prognosis than does the proliferative, and the presence of cavitation is always unfavourable.

**Treatment.** *Prophylactic:* Public health authorities are concerned with prophylaxis. Good housing and sanitation and a pure supply of milk are urgently needed. Isolation of all open cases would probably prove the most effective means of stamping out the disease. The milk given to infants up to the age of 2 years should be brought just to the boil as soon as it is received into the house. The tubercle bacillus in milk is destroyed by heat at 140° F. for 20 minutes and 194° F. for  $\frac{1}{2}$  minute. "Pasteurised" milk and "Certified" milk, which is tuberculin tested and pasteurised are safe, without further boiling, if the pasteurisation has been efficiently carried out. Fresh milk, either "Tuberculin tested" or "Accredited," cannot be guaranteed free from pathogenic organisms, and should not be used unless boiled. Prophylactic inoculation of infants with vaccines made from attenuated living bovine bacilli, such as the B.C.G., (*bacille-bilité Calmette-Guérin*), is practised extensively abroad, but is not devoid of danger. Open cases of tuberculosis in contact with children constitute a grave source of danger.

*Curative: Rest.* As soon as active pulmonary tuberculosis is diagnosed the patient should be put to bed and kept there for at least a month. If the temperature does not fall to normal in 2 weeks, he

should be put on "absolute rest" for 7 to 10 days. During this time, he lies flat, with only one pillow, and should not feed or wash himself, or read or get out of bed at all. Providing the temperature falls to normal the patient is gradually allowed to sit up in bed, then to lie on a couch for  $\frac{1}{2}$  to 2 hours a day, and later to dress and get up for 2 to 8 hours a day. Subsequently increasing amounts of walking exercise are taken, a record being kept of the temperature and pulse. If the temperature rises to  $99^{\circ}\text{F}$  any evening the patient should spend the next day in bed. *Fresh air.* The bedroom windows should be kept open day and night. The patient should be sufficiently well wrapped up to prevent shivering. He may lie in his bed on a balcony or verandah, or in a revolving shelter, but should not be exposed to rain, strong winds or direct sunlight. *Food.* The diet should be sufficient to restore the patient to his proper weight. This often means an increase of about a stone for an adult. A liberal mixed dietary should be given, and solid food may be eaten during the febrile period. No attempts at overfeeding should be made.

*Special Measures.* In early cases with a thin walled cavity, especially if situated peripherally in the lung artificial pneumothorax treatment should be attempted without the preliminary 4 weeks' bed treatment. Artificial pneumothorax treatment should also be tried if the disease is unilateral or mainly so as shown by X ray examination, in cases in which the temperature does not fall to normal after 3 to 4 weeks' rest in bed. Whether or not collapse is possible can only be determined by 3 or 4 trials at different sites as adhesions may prevent an adequate collapse. In some cases a bilateral pneumothorax will yield good results. The patients suffer very little dyspnoea when they are properly stabilised. In acute bronchopneumonic or pneumonic cases the artificial pneumothorax treatment should also be tried at once. It is not justifiable to wait for 3 or 4 weeks to see if the temperature will fall with rest. When a pneumothorax has been satisfactorily established it should be maintained for an indefinite period in the absence of complications. Oleothorax treatment consists in the injection of an oily substance such as Gomenol into the pleural cavity in cases of artificial pneumothorax. This is used either as a substitute for refills with air if a patient is going abroad to some place where such refills are unobtainable, or to try to prevent the lung from expanding in cases of "pneumothorax obliterans." There is a serious risk of the pleural contents becoming infected, necessitating rib resection, and for this reason such treatment is not recommended. If an artificial pneumothorax is not possible, or only produces a partial collapse owing to adhesions, and these adhesions cannot be divided with the use of a thoracoscope, a certain degree of collapse and rest for the affected lung can be obtained by the operation of phrenic crush or avulsion. In the latter the phrenic nerve is divided in the neck and pulled out from its thoracic attachments. The corresponding half of the diaphragm is paralysed and the diaphragm rises in a satisfactory case, about 2 inches. This allows both base and apex of the lung to collapse to a certain degree. A phrenic crush will cause temporary paralysis of the

diaphragm for about 6 months. If good results ensue the paralysis can be made permanent by avulsion of the nerve. Apicolysis is now rarely used to collapse an adherent apex with a deep-seated cavity. Paraffin wax is injected between the chest wall and parietal pleura. Extrapleural pneumothorax is an alternative procedure, but an upper stage thoracoplasty is now usually preferred. In extrapleural pneumothorax a portion of the fourth rib is resected posteriorly and the lung and adherent parietal pleura are stripped from the endothoracic fascia. In the space thus formed, refills of air are given with high pressures of + 18 cm. water or more. This operation has the advantage of not causing serious disturbance to the patient. Thoracoplasty is a severe operation. It may be justifiable in certain specially selected cases, in which the lesion is strictly unilateral, of a fibrotic type, and in which the lung is adherent to the chest wall, so that it cannot be collapsed by a pneumothorax. In the Sauerbruch operation the posterior portions of the first 10 or 11 ribs on the affected side are removed, usually in a three-stage operation, and the chest-wall thus falls in. The lateral collapse of the lung thus obtained is better than the antero-posterior. The Semb operation is now more often performed. The whole of the first three ribs are removed and the apex of the lung drops down when the supporting fascia is divided (extrafascial apicolysis). The lower ribs may be subsequently resected as in the Sauerbruch operation. The result is, of course, permanent, and if the other lung becomes affected the collapsed side cannot be allowed to re-expand, as can be done with an artificial pneumothorax. In cases of pulmonary tuberculosis in which the disease is unilateral or bilateral, and when the lung cannot be collapsed by an artificial pneumothorax, and the temperature will not settle to normal with rest in bed, the question of administration of Sanoecrysin (aur. et sod. thiosulphas) should be considered. This is a gold salt which is given intravenously. The initial dose should be small, 0.01 G., and if there is no reaction this is increased at intervals of 5 to 7 days in the following doses: 0.025 G., 0.05 G., 0.1 G., 0.25 G. and 0.5 G. It may not be possible to get above the 0.25 G. dose, owing to the reaction produced, but the total amount to be given in a course should be between 4 and 5 G. Reactions include: Fever, nausea, vomiting and diarrhoea. Albuminuria. Headache or meningeal symptoms. Cutaneous rashes and stomatitis. Enlargement of glands. Agranulocytosis. Owing to the risk of exfoliative dermatitis, it is not safe to give a subsequent injection if even a slight skin rash has been produced by a previous one. During and after a course of Sanoecrysin the patient should not expose the face to bright sunlight, as a permanent violet discoloration of the skin may ensue. If Sanoecrysin cannot be tolerated, or if the veins are difficult, Allochrysin can be injected intramuscularly, starting with 0.05 G., then 0.1 G., and finally 0.2 G. The latter dose is given every fifth day for 2 or 3 months. If these measures fail to lower the temperature, the outlook is very grave and the disease usually spreads and causes the death of the patient.

*Treatment of Complications.* In any case certain symptoms or complications may call for special treatment. The most important of these

are Cough If dry and ineffectual this may be checked by effort on the part of the patient, assisted by a sedative linctus such as Syr codein phosph, glycerin and succ. limonis,  $\overline{aa}$  partes  $\overline{equales}$  m 60 occasionally, or a lozenge, such as Ext glycyrrhiz gr 3, ol anisi m  $\frac{1}{2}$ , mass troch acac gr 10, or a mixture such as Tnc opu camph m 20, syr pruni serotin m 30 sp chlorof m 7, aquam ad fl oz  $\frac{1}{2}$  If there is much secretion expectoration can be helped by a hot water medicine such as Sod chlorid gr 3, sod. bicarb gr 5, sp chlorof m 5 aq anisi dest (B P C) ad fl oz 1 in an equal quantity of hot water Pain due to pleurisy can be treated by strapping the chest in a position of full expiration or by painting the affected part of the chest with liq iodi fort

Night sweats are at times very distressing The bed clothes should be light and the windows kept well open at night. A pill containing Zn oxid gr 2 and ext belladon sicc gr  $\frac{1}{2}$  at night should also be tried Paterson recommended that the patient should sleep on a grass mat placed between the sheet and mattress When the sweat occurs the patient should be sponged with toilet vinegar the night clothes changed and a warm drink given Hemoptysis The treatment is considered on p 178 Laryngitis The patient must neither speak nor whisper, and silence should be preserved for periods up to 6 months all communications being made by signs or by writing He must not smoke An inhalation of a few drops of a solution placed on the pad of a Burney Yeo mask every hour, may be used continuously while the patient is awake A solution frequently used is as follows Ol cassiae ol eucalypt ol abietis, creosoti  $\overline{aa}$  partes  $\overline{equales}$  If there is severe pain on swallowing a powder composed of equal parts of benzocaine and orthocaine may be used As much as will cover a sixpence is placed on the palm of the hand and inhaled directly into the larynx through a curved Leduc's glass tube, just before a meal In other cases, when the pain prevents swallowing even liquids, relief may be obtained by using a glass tube, which dips into the milk which is placed on the floor the patient sucking it up with his head hanging over the edge of the bed Firm pressure applied over both ears by the palms of the hands may also enable the patient to swallow in comparative comfort If these measures fail, alcohol injection into the superior laryngeal nerve may give temporary relief Diarrhoea This may result from irritation of food, from toxæmia and amyloid degeneration or from tuberculous ulceration of the intestines It is usually a sign of grave import The diet should be reduced and consist chiefly of milk Vitamins should be administered in the form of cod liver oil fl oz  $\frac{1}{2}$  and tomato juice 3 oz t d s Five mils of a 5% solution of hydrated calcium chloride should be given intravenously This should be repeated in 24 hours if the pain persists Pil plumbi cum opio gr 4 t d s may give relief, or a starch and opium enema (starch gr 60, water fl oz 2, and tnc opu m 30) can also be used.

The appetite may be improved by the use of a tonic such as Tnc. nuc. vom m 5, sod bicarb gr 10, sp chlorof m 7, infus gent co rec. ad fl oz.  $\frac{1}{2}$  Fl oz.  $\frac{1}{2}$  ex aqua t d s a c Extract of malt with cod-

liver oil m. 60 t.d.s. p.c. is useful for increasing weight, and Collosol Calcium m. 60 t.d.s. may be given by mouth periodically with the hope of aiding the healing process in the lungs.

*Sanatorium Treatment.* This is available for patients who have passed through the preliminary bed treatment, and have reached the stage of being up for 6 to 8 hours a day, without a febrile reaction. The chief advantages of the sanatorium are as follows: The patient is kept under skilled medical supervision during the difficult period of convalescence. He does not feel he is a nuisance or a danger to those at home. His immunity is increased by the *régime* of exercise and rest graduated according to each patient's needs. The rules of living which will be of the greatest value in enabling him to keep fit are inculcated. These are concerned with fresh air, food, exercise, rest, avoidance of alcohol and excessive smoking. Some patients are temperamentally unsuited for sanatorium life, they cannot stand the depressing effect of being continually surrounded by patients suffering from, and talking about, the same disease, and unless special arrangements are provided the life may be too rigorous for those past middle age or liable to bronchitis.

*Climatic Treatment.* Advice has to be given as to the best climate for the convalescent treatment, whether carried out in a sanatorium or elsewhere. During the initial bed treatment the patient is best placed where all the special forms of treatment described above are available, and where there are X-ray facilities. Subsequently he may go to various climatic resorts at home or abroad. It should be clearly understood that it is not necessary to go abroad to be cured of tuberculosis; further, if a patient does go abroad one winter he is apt to think that he is running a grave risk if he stays in England subsequently in the winter for the first 2 or 3 years. The various climates may be grouped as follows: Mountain resorts. These usually imply Switzerland at a height of about 5,000 feet. Such resorts are especially suitable for early afebrile patients, who are free from such complications as bronchitis, laryngitis, heart disease, arteriosclerosis, nephritis, emphysema and insomnia. Patients with active and extensive disease should not be sent to Switzerland. Marine climates are available in England at such places as Ventnor and Bournemouth. They are especially valuable in cases of pulmonary tuberculosis complicated by laryngitis or bronchitis. Inland climates include moorlands and pine country. They are usually at a moderate altitude of a few hundred feet and are suitable for all classes of pulmonary tuberculosis who will benefit by sanatorium treatment. Sea-voyages are always contraindicated in tuberculosis, and should be absolutely vetoed.

*The After-treatment.* When arrest of the disease has been firmly established, the question of return to work and the suitability of the employment has to be decided. In general all heavy muscular work should be avoided, and outdoor occupations are unsuitable unless the patient is protected from the extremes of climate and from getting wet. Indoor occupation is not harmful provided the environment is healthy and the patient can look after himself adequately at home. There is

less probability of financial worry if the patient returns to the occupation for which he has been trained than if he embarks upon a fresh one. The after-care of tuberculous patients in special colonies, in which they can live with their families under medical supervision, is still in the experimental stage, but holds out encouraging hopes. There remain a large number of patients who do not do well with hospital or sanatorium treatment. These are often sent back to their homes, eventually to die. They are "open" cases of tuberculosis and undoubtedly are a great risk to others. If all "open" cases of pulmonary tuberculosis were isolated the hope of stamping out this disease would be increased.

### Emphysema

**Definition.** Dilatation of the pulmonary alveoli.

**Varieties.** Certain varieties of emphysema are described which will be considered separately. These are: *Acute emphysema*, which is *vesicular or interstitial*. *Chronic emphysema*, which is *hypertrophic or atrophic*.

#### Acute Vesicular Emphysema

**Definition.** Sudden over distention of the pulmonary air vesicles.

**Etiology.** Acute vesicular emphysema is a rare condition associated with severe coughing or muscular strain. It may be met with in whooping-cough, asthma or asphyxia, and in children is often revealed radiologically adjacent to a consolidated area of lung in lobar pneumonia.

**Clinical Findings.** The symptoms which might be associated with it are masked by those of the primary condition. Its presence can often be detected radiologically by a ring shadow.

#### Acute Interstitial Emphysema

**Definition.** A condition characterised by the presence of air in the interstitial tissues of the lungs, the air is derived from ruptured pulmonary alveoli.

**Etiology.** The air vesicles may rupture with comparatively moderate muscular exertion, such as while playing a game of hockey. Other causes include a wound of the chest, fractured rib, or the severe paroxysms of cough in whooping cough or bronchopneumonia. It occurs occasionally in pulmonary tuberculosis, apart from any effort.

**Pathology.** The escaped air tracks along the pulmonary roots and may reach the mediastinum, or appear as surgical or subcutaneous emphysema in the neck and chest.

**Clinical Findings.** In cases occurring during exercise, the patient feels a tightness or pain in the neck and chest, and shortness of breath.

**On Examination.** The crackling signs of surgical emphysema may be felt on the chest or neck. If the air has extended into the anterior mediastinum the superficial cardiac dulness is obliterated, the heart sounds are distant, and crackling sounds may be heard over the præcordium as the patient breathes and the heart beats.

**Course.** The air is usually absorbed spontaneously in a few days.

**Treatment.** The patient is kept in bed. He usually prefers to be



propped up. A hypodermic injection of morphin. sulph. gr. 1/6 will help to check cough and induce sleep.

### Generalised Hypertrophic Emphysema

#### (*Substantive or Idiopathic Emphysema*)

**Definition.** A condition of generalised dilatation of the alveoli, with an insidious onset.

**Etiology.** The cause is unknown. It is most probably due to a constitutional weakness of the elastic tissue of the lungs, associated with some degree of bronchitis. Chronic alcoholism may also play a part. The various theories propounded to account for its etiology include : 1. Excessive inspiratory efforts : In chronic bronchitis and asthma the inspiratory efforts are stronger than the expiratory, hence the lungs become over-distended. 2. Violent expiratory efforts : These may be associated with chronic cough, asthma and with occupations such as playing wind instruments or lifting heavy weights. 3. Premature ossification of the costal cartilages : The chest wall might become fixed in an inspiratory position, with secondary emphysema of the lungs. Clinically the chest wall is often unduly rigid in emphysema.

**Pathology.** Both lungs are affected. At autopsy they are bulky, pale and soft, and lacking in elasticity. Bullæ may be seen on the surface, especially along the anterior borders. On section the lungs are pale and the dilated alveoli may be noticeable. They float in water. As the alveoli dilate, the dividing septa break down and bullæ form. Aeration of the blood is diminished in emphysema for the following reasons : The aerating surface is reduced owing to the loss of the septa. The lumen of the vessels is narrowed in the stretched alveolar walls, thrombosis occurs in the pulmonary arterioles, and so circulation is impeded. Some blood is short-circuited from the pulmonary arterioles to the bronchial venules. The right side of the heart dilates and the motor power of the pulmonary circulation is reduced.

**Clinical Findings.** The patient is usually a male, of middle age or over. He may give a history of winter cough or of asthma. His chief complaint is shortness of breath on exertion.

**On Examination :** The patient may be cyanosed, slightly or to an extreme degree, and the fingers may be clubbed. The chest. Inspection : The diameter is increased antero-posteriorly (barrel-shaped chest), the ribs run more transversely than normal, the subcostal angle is wide and there is kyphosis in the upper dorsal region. Post-mortem and X-ray examinations have shown that marked emphysema may occur without the presence of the "barrel-shaped syndrome" and *vice versa*. Expansion is poor, the movement on inspiration being chiefly one of elevation. Dilated venules may be seen along the line of the diaphragmatic attachment. Palpation : Tactile fremitus is diminished. The cardiac apex cannot be felt. Percussion : The note is hyperresonant generally. The areas of cardiac and hepatic dullness are reduced. Auscultation : The breath sounds are weak. Expiration may be prolonged ; scattered rhonchi or râles may be heard. There is no bronchophony and no whispering pectoriloquy. X-ray examination :

The translucency of the lungs is increased, especially at the bases. The ribs run more transversely than normal. The diaphragm is low and shows diminished movement. The heart shadow is small, as the heart rotates to the right with the depression of the diaphragm. The vital capacity. This is determined by the use of a spirometer. A normal adult can expire about 3,600 c.c. of air after a deep inspiration. In emphysema this volume may be reduced by a half or more. The sputum. This is scanty and rather frothy. The blood may show an excess of red cells.

**Differential Diagnosis** The diagnosis of emphysema, which is often made with such certainty clinically, has been shaken by post-mortem observations in which no such condition has been found to exist. The X-ray findings are most reliable.

**Course and Complications** Emphysema tends to be progressive. Chronic bronchitis, asthma, dilatation of the right side of the heart, *tricuspid regurgitation*, a pulsating liver and other signs of heart failure are liable to occur (see Cor pulmonale, p. 223). Other complications include pulmonary tuberculosis, spontaneous pneumothorax or acute interstitial emphysema, due to rupture of a bulla.

**Prognosis** There is usually no immediate danger, apart from the presence of heart failure. The ultimate outlook is very unfavourable. The ability to adopt a suitable mode of life very materially improves the prognosis.

**Treatment** *Prophylactic* Occupations involving strain should be avoided by those predisposed to, or with early signs of emphysema.

*Curative* There is no curative treatment. All that can be hoped is to decelerate the natural progress of the disease. If possible the patient should winter out of Europe, as in South Africa or Egypt. If this is not feasible, every effort should be made to avoid bronchitis, and if it occurs it should be treated early and rigorously. In severe cases a course of compressed air baths may be given. The patient enters a closed chamber, and the pressure within is gradually raised to that of  $1\frac{1}{2}$  atmospheres. After half an hour it is slowly lowered to normal again. The whole process takes about  $1\frac{1}{2}$  hours. These baths can be given two or three times a week, if the patient derives benefit from them. Heart failure must be treated by rest, venesection and digitalis (see p. 227).

#### *Localised Hypertrophic Emphysema* (Compensatory or Secondary Emphysema)

**Definition** Dilatation of the alveoli in localised areas of the lungs.

**Etiology** Compensatory emphysema is a secondary process around areas of consolidated, fibrosed or collapsed lung. It is thus associated with pneumonia or bronchiopneumonia, new growths, fibrosis, bronchitis and pleural effusion.

**Pathology** Inspiratory efforts, associated with an obstruction to the air entry of a localised area of lung, will lead to alveolar dilatation of neighbouring parts of the lung. If one lung is largely put out of action, as by tuberculous fibrosis, compensatory emphysema will

occur in the other. There is no true hypertrophy of lung tissue, but changes similar to those described on p. 161 are seen. The process therefore diminishes the aerating power of the lung.

**Clinical Findings.** The symptoms are usually those of the primary condition, with perhaps some additional dyspnoea due to the emphysema.

**On Examination:** There are not usually any abnormal signs directly attributable to the emphysema unless a lobe, or the greater part of a lobe, of the lung is involved. In such cases the emphysematous side of the chest is hyperresonant, the hyperresonance may extend across the mid-line of the sternum, and in the early stages the breath sounds are loud and exaggerated. After the condition has persisted for some time, the air entry becomes weaker and expiration is prolonged.

The condition calls for no special treatment.

### Atrophic Emphysema

#### (*Senile Emphysema*)

**Definition.** Enlargement of the pulmonary alveoli, due to degeneration of their septa.

**Etiology.** Atrophic emphysema is a variety of senile degeneration.

**Pathology.** The lungs, as seen at autopsy, are small, dark and friable. Small bullæ may be present on the surface, and, on section, the enlarged alveolar spaces are seen.

**Clinical Findings.** The patient, who is usually over the age of 60, complains of progressively increasing shortness of breath on exertion, and usually he has cough and expectoration due to associated bronchitis.

**On Examination:** The patient is usually thin and wasted. The chest is flat and expansion is poor. The tactile fremitus is diminished. The percussion note is hyperresonant, but the cardiac and hepatic dulness are not diminished to any degree. The breath sounds are weak and expiration is a little prolonged. Adventitious sounds due to bronchitis may be heard.

**Treatment.** No special treatment is available beyond the care necessary for an elderly patient who is liable to bronchitis or to dilatation of the heart.

### Tumours of the Lungs

**Simple Tumours.** These usually arise in the bronchi and have been considered on p. 135.

**Malignant Tumours.** *Primary carcinoma of the lungs* arises in a bronchus and is described on p. 130. Sarcoma or endothelioma may be primary or secondary, whereas hypernephroma, malignant deciduoma and seminoma are secondary tumours. *Primary sarcoma.* This is a rare, solitary and rapidly growing tumour occurring in early life. *Secondary sarcoma.* The primary growth is in bone or in an melanotic tumour. *Secondary lymphosarcoma.* In addition to the growth in the lungs enlarged glands are usually found elsewhere, as in the neck and axillæ, and diagnosis is made by gland section. *Secondary teratoma and seminoma.* The primary growth is in the testicle, the secondary

growths in the lungs are described as "cannon ball" tumours on X ray examination

### Fibrosis of the Lungs

(including *Chronic Interstitial Pneumonia*)

**Definition** An excess of fibrous tissue in the lungs

**Etiology** The fibrosis is usually productive in origin, being formed in response to irritation, less frequently areas of replacement fibrosis occur

**Pathology** The fibrous tissue may be distributed in various ways in the lungs. It may be diffuse, as in tuberculosis, pneumokoniosis, and in chronic passive hyperæmia. Lobar fibrosis may follow lobar or bronchopneumonia. Peribronchial fibrosis is met with in chronic bronchitis. Localised fibrosis occurs around a tumour, cyst, abscess, or granuloma of the lungs, and after bronchopneumonia. Pleurogenous fibrosis is secondary to chronic pleurisy, fibrous tissue extending into the subjacent lung. When the fibrosis is localised a depressed thickened area may be seen and felt. This is often found at the apex of a lung due to a healed tuberculous focus. With diffuse fibrosis the lung is contracted, firm, and darker than normal. There are often pleural adhesions. On section the lung is tough, the strands of fibrous tissue are apparent and there may be bronchial dilatation.

**Clinical Findings** It is only in the diffuse variety that symptoms are likely to be noted. The patient complains usually of shortness of breath on exertion, with cough and expectoration, especially in the winter. Occasionally unilateral fibrosis will cause dysphagia by torsion on the œsophagus.

**On Examination** In a long standing case there is usually some cyanosis of the face and clubbing of the fingers. If the fibrosis is unilateral there will be flattening and diminished expansion on one side of the chest, and the cardiac impulse may be seen displaced towards the affected side. The corresponding shoulder may be low, and some scoliosis present. If the fibrosis affects the left upper lobe, cardiac pulsation is usually seen in the second and third left intercostal spaces, close to the sternum due to uncovering of the heart. **Palpation** The diminished expansion is confirmed and tactile fremitus is lessened. **Percussion** The note is impaired over the affected lung, and it may be hyperresonant on the other side. **Auscultation** The breath sounds are weak over the fibrosed lung, and harsh on the opposite side. Some leathery fibroid râles may be heard, and frequently rhonchi are present in both lungs. Vocal resonance is diminished, and there is no whispering pectoriloquy. If areas of consolidation or excavation are present, the breath sounds are more bronchial in type and voice conduction is increased. Basal fibrosis is often seen in children after bronchopneumonia. The heart is slightly displaced to the affected side, the note at one base is impaired, the air entry weak, and fine to medium râles are heard constantly on deep breathing or after cough.

**Differential Diagnosis** The diagnosis of pulmonary fibrosis does not usually present difficulties, but it may be impossible to be certain

as to its causation. A Wassermann test should be done, and, if positive, the effect of a course of potassium iodide and mercury (see p. 43) determined. The sputum should be examined several times for tubercle bacilli, and the nature of the predominating organisms investigated. The occupational history of the patient and the X-ray findings may point definitely to pneumokoniosis. Fibrosis of a lower lobe is often the result of pneumonia, bronchopneumonia or pleurisy. This is especially liable to occur in children after bronchopneumonia associated with measles or whooping-cough.

**Course and Complications.** Fibrosis is usually slowly progressive. It may lead to bronchiectasis, and be accompanied by areas of compensatory emphysema.

**Prognosis.** In tuberculosis, fibrosis is welcomed as an indication of arrest of the disease. Apart from this, fibrosis limits the functions of the lungs and increases the strain on the heart, and so tends to shorten life. It may also conduce to bronchiectasis.

**Treatment.** *Prophylactic:* Adequate inspiratory breathing exercises should be done during convalescence in every case of bronchopneumonia, pneumonia and empyema, to expand the base of the lung and prevent fibrosis.

*Curative:* In basal fibrosis, non-tuberculous in origin, inspiratory breathing exercises should be performed daily, to expand the lung and endeavour to prevent the development of bronchiectasis. Dusty environments should be avoided. Iodides should be given, as mentioned above, if it is considered probable that the fibrosis is syphilitic in origin. The patient must live well within the limits of his respiratory and cardiac reserves, and climatic treatment in the winter is of value in preventing bronchitis.

### Pneumokoniosis

#### (Dust Disease of the Lungs)

**Definition.** Fibrosis of the lungs, due to inhalation of dust.

**Etiology.** The following varieties are described, according to the nature of the irritant: Anthracosis (coal). Silicosis (silica as in the following occupations:—Gold, tin, zinc or hematite iron ore, and coal mining; sandblasting; flint and pebble crushing; the manufacture of abrasive soaps; metal grinding; slate quarrying; granite, sandstone and pottery work). Lithosis or chalicosis (stone particles). Siderosis (tin, copper, lead or iron), this is probably silico-siderosis. Asbestosis (metallic particles containing silica and iron, and also vegetable fibres). Asbestos workers are employed in making matches, filters, paints, roofing tiles, brake linings, etc. Byssinosis (cotton particles).

**Pathology.** When particles of dust are inhaled for long periods, fibrotic and bronchitic changes ensue in the lungs. It is very doubtful whether in man the particles reach the lungs from the alimentary tract and the lymphatic system. Coal-dust particles are to a certain extent expectorated, but silica particles tend to remain in the lungs, and pulmonary tuberculosis is very prone to supervene. Tuberculosis is rare in coal-miners, but common in gold-miners, and it also occurs in

some cases of asbestosis. Dusts containing free crystalline silica ( $\text{SiO}_2$ ) are dangerous, and their inhalation leads to fibrosis and tuberculosis. Silicosis is not uncommonly combined with anthracosis in coal miners' lungs, but here it leads to bronchitis rather than to tuberculosis. The reason that tuberculosis is rare in coal miners may be that finely divided charcoal has the power of adsorbing tuberculin. In anthracosis the lungs, as seen at autopsy, are black, in silicosis they are grey, and in siderosis they are brown. They are firm and contracted, due to nodular or diffuse fibrosis, and the pleura is thickened with adhesions. The bronchi are often dilated and the root glands are enlarged, hard and pigmented. In asbestosis the silica is combined with such bases as magnesium, iron, calcium, sodium and aluminium, and the silicate so formed is less dangerous than silica.

**Clinical Findings** The patient is usually a male between the ages of 20 and 40, who gives a history of having worked for some years (usually over 6) in one of the dusty occupations mentioned above. He complains of cough and expectoration, with shortness of breath, and later of lassitude and loss of weight.

**On Examination** In the early stages the only abnormalities are those detected by the X rays. In silicosis small nodules of fibrous tissue may be seen in the lung fields, with diffuse streakiness of the lungs and heavy root shadows. The nodular shadows become more obvious as the condition progresses, and later a diffuse mottling occurs, chiefly in the central zones of the lungs. In asbestosis there is a reticular fibrosis. The physical signs in an established case are chiefly those of chronic bronchitis and emphysema, the air entry over both lungs being weak. The sputum in asbestosis may contain microscopical golden yellow "asbestosis bodies," with bulbous extremities, consisting of a central core of asbestos surrounded by discs which give the Prussian blue reaction for iron, and are probably composed of iron silicate.

Some cases of acute silicosis have occurred in workers in a factory manufacturing a cleaning powder containing ground silica, sodium carbonate and soap. The alkali is probably responsible for the rapid onset of the symptoms. Death occurred in two cases, and in one of them tuberculous lesions were also demonstrated in the lungs. Cases have also been described in tunnellers and in rock drillers in lead mines.

**Differential Diagnosis** The diagnosis of silicosis can usually only be established with certainty by X ray examination of the lungs.

**Course and Complications** The course is progressive, unless arrested by removal of the patient from the noxious environment. Complications include bronchitis, bronchiectasis, emphysema, tuberculosis and cardiac dilatation. Silicosis is occasionally followed by carcinoma of a bronchus. There is not sufficient evidence to say whether asbestosis predisposes to tuberculosis, although it undoubtedly occurs in certain cases.

**Prognosis** This is unfavourable unless the patient is removed from his dusty occupation in the early stages of the disease.

**Treatment** *Prophylactic* Measures should be taken in all dusty occupations to prevent inhalation of the dust. These include the use of

respirators, sprays and fans. The workers should be examined and X-rayed every 6 months.

*Curative*: The patient should be removed from his occupation. The treatment then is as for chronic bronchitis and emphysema.

### Syphilis of the Lungs

The lungs may be affected in congenital or acquired syphilis.

*Pathology.* In congenital syphilis a condition of "white pneumonia" is usually present. A lobe, or the greater part of one or both lungs, may be involved. The affected portion is enlarged, and depressions caused by the pressure of the ribs may be seen on the surface. The lung appears pale and dry and sinks in water. On section the lung may somewhat resemble the tissue of the pancreas ("pancreatisation"). The alveolar epithelium is degenerated, and the alveoli contain some leucocytes. There is round-celled infiltration of the interalveolar tissue with numerous small gummata. The *Treponema pallidum* is present. In acquired syphilis the lesion may take the form of a gumma, or of a peribronchial and perivascular fibrosis.

*Clinical Findings.* The infant with congenital syphilis of the lungs is still-born, and the lesion is demonstrated at autopsy, or death occurs after a few days, during which signs of pulmonary consolidation may have been detected. The Wassermann reaction of the blood, both of the mother and child, is generally positive, and other stigmata of congenital syphilis (see p. 367) are noted. In adults the clinical findings may be those of diffuse pulmonary fibrosis (see p. 164), of a local pulmonary tumour, or of a blocked bronchus if the gumma is situated intrabronchially. In the latter case the lesion is demonstrated by finding clinical evidence of collapse of a portion of the lung (see p. 171), by the X-ray appearance after Lipiodol injection, in which the Lipiodol fails to enter the collapsed area, and finally by bronchoscopy in which the site of the obstruction can be visualised and a portion of the granuloma removed for examination.

*Differential Diagnosis.* This may be very difficult in cases of acquired syphilis. Other causes of pulmonary fibrosis, especially tuberculosis, require to be eliminated. Further causes of bronchial obstruction, such as a new growth or pressure from an aneurysm may be misleading. The positive Wassermann reaction and the response to anti-syphilitic treatment, as judged by the clinical and X-ray evidence, are of paramount value.

*Course and Complications.* Syphilis of the lungs pursues a slowly progressive course. Complications include bronchitis, bronchiectasis and abscess of the lungs. The abscess may discharge pus and gummatous material through the chest wall.

*Prognosis.* This is favourable in the acquired variety, provided that an early diagnosis is made and adequate treatment is given before complications occur. In advanced cases, where there is marked bronchial obstruction, with retention of secretion distal to the stricture, the results obtained are very disappointing.

**Treatment** A preliminary course of neoarsphenamine should be given, beginning with 0.3 G (see p. 570). After six injections a course of potassium iodide, in doses increasing from gr. 5 to gr. 30 or 60 t.d.s. with liq. hydrarg. perchlor. m. 20, t.d.s. should be given for 2 to 3 months. The results should be checked by X-ray examination.

### Actinomycosis of the Lungs

**Etiology** The causative organism is the *Actinomyces bovis* (*Streptothrix actinomyces* or ray fungus). This may gain access to the lungs from the teeth or tonsils (see p. 585).

**Pathology** Granulomatous lesions are found in the lungs, often in the lower lobes. Suppuration and fibrosis occur around them, and they may spread to involve the pleura, chest wall or liver.

**Clinical Findings** The history of the case may closely resemble that of chronic pulmonary tuberculosis or of a neoplasm of the lungs. Thus the patient complains of cough, expectoration which may be bloodstained, progressive loss of weight and irregular fever.

**On Examination** The physical signs in the lungs are very variable. Thus they may be those characteristic of bronchitis, or of an area of consolidation or excavation of the lung. The lesion may ulcerate through the chest wall, producing a purulent discharge containing "sulphur" granules. The sputum should be specially examined for the ray fungus, by the appropriate staining methods.

**Differential Diagnosis** This may be very difficult before the organism is found in the sputum. The X-ray findings often closely resemble those of chronic pulmonary tuberculosis or of carcinoma of the lung.

**Course and Complications** The course is usually slowly progressive, the infection may spread elsewhere, especially to the liver or chest wall.

**Prognosis** The outlook is unfavourable.

**Treatment** Sulphapyridine (M. & B. 693) should be administered, 1 G t.d.s. for 6 days, followed by a second course 10 days later. If this is not successful potassium iodide should be given in increasing doses, beginning with gr. 5 t.d.s. and working up to gr. 60 or 90 t.d.s. Cutaneous sinuses should be injected from time to time with Lipiodol or Neo Hydriol (ol. iodisat B.P. Add.) and treated with deep X-rays or a radium pack.

### Aspergillosis of the Lungs

**Etiology** Infection with a fungus, the *Aspergillus fumigatus*. The infection is spread by grain.

**Clinical Findings.** The patient is usually a miller, farm worker or pigeon breeder. He complains of symptoms resembling those of chronic pulmonary tuberculosis, more rarely he is acutely ill.

The diagnosis can only be made if the organism is found in the sputum.

**Treatment.** This consists in administering potassium iodide, as for actinomycosis (see above).



### Moniliasis of the Lungs

**Etiology.** The causative organisms are various fungi of the monilia group. It is met with among tea tasters in Ceylon.

**Clinical Findings.** The patient complains of symptoms resembling those of chronic bronchitis, and the causative organisms are found in the sputum.

**Treatment.** This consists in the administration of potassium iodide, in gradually increasing doses from gr. 5 to gr. 60 t.d.s.

### Collapse of the Lungs

**Varieties.** Collapse of the lungs may be congenital or acquired. In congenital collapse (atelectasis) the lung or a portion of the lung has never expanded. Acquired collapse (apneumotosis) may be active or passive.

#### Congenital Pulmonary Collapse

This is met with in still-born infants or in those in whom the respiratory efforts are very feeble, or where there is obstruction to the airway.

#### Active Lobar Collapse

##### (Massive Collapse)

**Etiology.** Massive collapse of the lungs is met with under a variety of conditions, such as injury to the chest which does not necessarily damage the lung, injury to the abdomen or legs, abdominal operations especially on the upper abdomen and when the bandages are tightly applied preventing adequate expansion of the lower lobes of the lungs, operations on the thyroid, post-diphtheritic paralysis, anterior poliomyelitis, Lipiodol injection into healthy bronchi, hæmoptysis, etc.

**Pathology.** Various theories have been propounded to account for the collapse. These include: 1. Neuritis of the phrenic nerve or a reflex inhibition of the diaphragm. As, however, avulsion of the phrenic nerve is not followed by massive collapse of the lung, this view does not appear tenable. 2. A reflex inhibition of the diaphragm and intercostal muscles. This may account for some cases, as when there is a non-penetrating wound of the opposite side of the chest. 3. Reflex constriction of the bronchioles with inspiration, expiration being normal, so that the lung becomes deflated. Such a reflex might be initiated from stimuli in the chest wall or other part of the body, or from intra-bronchial causes, such as blood clot. 4. Plugging of the bronchi with mucus or blood clot, the retained air in the alveoli being absorbed into the blood stream. This appears to be the explanation in the majority of cases. The fact that collapse can occur in one lung when there is a trivial injury to the chest wall on the opposite side is opposed to the view that it is due to exudative bronchial obstruction, and its appearance within a few minutes of a Lipiodol injection favours the view that it is due, at any rate in this instance, to reflex spasm of the bronchioles. The affected lung is small, blue, and sinks in water. The mediastinum is displaced to the affected side.

**Clinical Findings.** The usual history in civil life is that about two days after an operation, such as on the gall-bladder, appendix or thyroid gland, the patient is suddenly seized with dyspnoea and pain in the lower part of the chest. He is also collapsed. Post-operative massive collapse does not occur so frequently as formerly, possibly owing to improvements in pre operative technique and in the administration of the anæsthetics.

**On Examination:** The patient is cyanosed in some instances. In the early stages the affected side of the chest is immobile and the air entry is very weak over the affected lung, usually over the lower lobe. The heart is displaced to the affected side. The temperature, and pulse and respiration rates are raised. Later, there is dulness over the affected lobe, with bronchial or tubular breathing, whispering pectoriloquy and bronchophony and a few fine râles. The breath sounds over the opposite lung are harsh, and the percussion note may be hyperresonant. There is usually some frothy expectoration. X-ray examination shows displacement of the mediastinum to the affected side with elevation of the diaphragm. The ribs become approximated, giving a tiled appearance. The collapsed lung casts a shadow which may be sufficiently dense to obscure the ribs.

**Differential Diagnosis.** Before massive collapse was clearly described by Pasteur at the Middlesex Hospital, London, in 1908, it was usually mistaken for post-operative pneumonia. The chief distinguishing features are the homolateral cardiac displacement, the absence of rusty sputum and labial herpes, and the course of the illness, which is more rapid than that of pneumonia. The diagnosis is confirmed by X-ray examination, by which the collapsed part of the lung and the raised cupola of the diaphragm are revealed. The chief condition which has to be excluded is, in left-sided affections, a spontaneous pneumothorax, as the stomach resonance with a massive collapse may extend up as high as the fourth rib. This gives a hyperresonant note which may be thought to be due to air in the pleura. The displacement of the heart to the affected side serves, however, to exclude a pneumothorax.

**Course and Complications.** In about 2 or 3 days the affected lung re-expands, the dyspnoea ceases and the temperature, and pulse and respiration rates fall to normal. Pleurisy, bronchitis and pneumonia may occur as complications.

**Prognosis.** This is usually good, unless the collapse is bilateral or due to poliomyelitis, or unless pneumonia supervenes.

**Treatment. Prophylactic.** This consists in the administration of atropine before operations, and encouraging the patient to take periodically a few deep breaths after the operation. The inhalation of 5% carbon dioxide in 95% oxygen for a few minutes at the end of an anæsthetic assists in ventilating the lungs. Changes of position in bed and the avoidance of binders which constrict the lower ribs are also valuable.

**Curative:** The general collapse of the patient rather than the local collapse of the lung calls for treatment. Stimulants, such as strychnin.

hydrochlor. gr. 1/30 with atropin. sulph. gr. 1/100 should be given hypodermically and repeated in 6 hours if required. Bandages restricting the lower part of the chest should be loosened. The patient should inhale through a nasal catheter a mixture of oxygen and 5% carbon dioxide for several hours. Cardiac and respiratory stimulants such as Cardiazol (leptazolium, B.P. Add.) 1 mil. and Coramine (nikethamidum, B.P. Add.) 1.5 mil. should also be given subcutaneously every 6 hours. The introduction of 100 to 200 c.c. of air into the pleura, by means of a pneumothorax apparatus, affords relief by raising the intrapleural pressure, which is below normal. If these measures fail a bronchoscopic aspiration may be performed to remove any mucus present.

### Passive Collapse of the Lungs

**Etiology.** Passive collapse of the lungs may be due to : 1. Pressure outside the lung, as in pneumothorax, pleural or pericardial effusions, or after a phrenic avulsion or thoracoplasty operation. 2. Bronchial obstruction. This may be intra- or extra-bronchial. A foreign body, such as an acorn, may lodge in the main bronchus, or the lumen may be obstructed by plugs of mucus. Other causes include a new growth or gumma of the bronchus, the pressure of an aneurysm, fibrosis, or consolidation of the lung.

**Clinical Findings.** Unless the collapse is extensive and rapidly produced, and the pleural pressure is raised above zero, clinical symptoms are not usually noted. Thus the whole of one lung may be slowly put out of action by an artificial pneumothorax, without the patient experiencing any undue dyspnoea, and a patient may breathe comfortably in bed with both lungs almost completely collapsed by a bilateral artificial pneumothorax. The signs of local areas of collapse are dulness and weak air entry. These are often met with in cases of influenza and change rapidly from day to day, as the collapsed portion of lung re-expands and another zone collapses. In some cases collapse of the right middle lobe is present, which is revealed by the typical X-ray appearance in a lateral view, a triangular shadow being seen. Re-expansion can often be effected by breathing a mixture of 7% carbon dioxide and 93% oxygen delivered from a cylinder through a gas-bag and face mask for periods of 5 minutes daily. Inspiratory breathing exercises should also be practised. In other cases bronchoscopic aspiration is required.

### Hyperæmia of the Lungs

#### (Congestion of the Lungs)

**Definition.** Dilatation of the pulmonary arterioles and capillaries. There are two varieties of congestion of the lungs, active and passive.

#### Active Hyperæmia

This is an inflammatory condition in which the pulmonary and bronchial arterioles are actively dilated. The predisposing causes are inflammatory lesions of the lungs, bronchi or pleura. Obstruction of

the circulation in one part of the lung, as by an infarct, may cause collateral or fluxionary hyperæmia in other parts of the same or the opposite lung

**Clinical Findings.** The physical signs of active hyperæmia are slight dulness, weak breath sounds and râles over a localised area of the lung. There may be cough and expectoration, and the latter may contain bright blood. The so called abortive pneumonia (*maladie de Woillez*) is probably an example of active hyperæmia. Here the onset resembles that of lobar pneumonia, but the temperature falls in 24 to 36 hours and the patient improves. In the first stage of lobar pneumonia there is also active hyperæmia

### Passive Hyperæmia

Obstruction to the outflow of blood from the lungs leads to congestion of the capillaries. This may result from heart failure, as in mitral stenosis, auricular fibrillation, myocardial degeneration and pericardial effusion. Intrapulmonary causes include chronic bronchitis and emphysema, and fibrosis. The pulmonary veins may be obstructed by thrombosis or by the pressure of enlarged mediastinal glands. Hypostatic congestion is seen in elderly and bed ridden patients, owing to feeble action of the cardiac and respiratory muscles. If the hyperæmia is of some standing the lungs undergo "brown induration". There is often, in addition, œdema of the lung parenchyma. Hypostatic pneumonia results from an infection supervening in an area of hypostatic congestion.

**Clinical Findings.** The patient may complain of shortness of breath, cough and expectoration, but the symptoms and signs are largely those of the causative condition.

**On Examination.** There is some dulness at the base of one or both lungs, with diminished tactile fremitus, weak breath sounds and fine râles heard especially when the patient takes a deep breath. The latter may be very fine, of the nature of crepitations, indicating the presence of œdema. The red cell count may be increased to about 7 millions.

**Treatment.** *Prophylactic.* Hypostatic congestion in elderly patients may be prevented in some cases by confining the patient to bed as little as possible, and when in bed encouraging the patient to sit propped up and to take deep breaths from time to time.

*Curative.* This must depend upon the underlying cause. Venesection of 10 to 15 oz. may produce great amelioration of the congestion.

### Hæmorrhagic Concussion of the Lungs

#### (Blast Injury of the Lungs)

**Etiology.** Hæmorrhagic lesions are often found in the lungs, especially in children, from peace time injuries such as road accidents, in which no injury to the chest wall and no fracture of the ribs can be found. Blast from a high explosive bomb may kill without signs of external injury.

**Pathology.** Hæmorrhagic areas are found usually deep in the

lungs, and often at the costo-phrenic sinus, when the spleen or liver is also damaged. Blood-stained mucus may be present in the upper respiratory passages. There may also be hæmothorax and effusion of blood into the mediastinum.

**Clinical Findings.** The patient may be found dead as described above or complain of faintness, shortness of breath and pains in the chest or abdomen. *On Examination:* Patients are seen to be suffering from varying degrees of shock; there is pallor, cyanosis, a rapid and feeble pulse and low blood pressure. In addition there is dyspnœa chiefly of an expiratory type, the chest being over-distended and bulging. The physical signs in the lungs vary, the breath sounds are often weak throughout both lungs and a sudden rise of temperature may herald the development of a lobar or lobular consolidation. Abdominal pain, tenderness and muscular rigidity are met with in some cases, suggesting an acute abdominal condition. Hæmoptysis of varying degrees may occur, and restlessness is a predominant feature in all cases of blast injuries to the lungs. Ruptured ear drums are to be expected if the person has been sufficiently close to the explosion to suffer internal injuries from blast.

**Treatment.** Shock should be combated by placing the patient in a bed heated with hot bottles or an electric cradle. Morphine, sulph. gr.  $\frac{1}{2}$  should be given subcutaneously to control restlessness and severe pain, and, if necessary, repeated up to a maximum of gr. 1 in 24 hours. Oxygen should be inhaled from a B.L.B. mask or nasal catheter (see p. 143). Plasma transfusions of 1 to 2 pints are often helpful, but whole blood transfusions are only required if there is anaemia.

### Acute Œdema of the Lungs

**Definition.** A condition in which the pulmonary alveoli are suddenly flooded with serous exudate.

**Etiology.** The cause is not known. Probably there are several varieties. There are three main views. 1. The cardiac theory: The œdema is considered to be a manifestation of sudden left-sided heart failure, perhaps due to a disproportion between the effective power of the ventricles, the left expelling less blood than the right. 2. The toxic theory: This may account for certain cases met with in connection with chronic nephritis, pregnancy and infectious diseases, in which no cardio-vascular lesion is demonstrable. 3. The angio-neurotic theory: This helps to explain the sudden onset of the condition in young people, apparently in perfect health, and is supported by the simultaneous appearance of œdema of the face.

Clinically, acute œdema of the lungs is most often associated with arteriosclerosis, aortic disease, myocardial degeneration, coronary occlusion and chronic nephritis. Less frequently it occurs as a complication of aspiration of a pleural effusion.

**Clinical Findings.** The patient is usually a woman over the age of 40. She is apparently in normal health, when she is suddenly seized with faintness and apprehension; she then becomes very short of

breath and may rapidly pass into a stage of semi- or complete unconsciousness

**On Examination** If the patient is conscious she is found sitting up and alarmed, with great distress of breathing. The skin is pale, and moist, and slight cyanosis is present. The heart is usually regular, but rapid. Rales are heard all over both lungs. After a period varying from a few minutes to half an hour frothy fluid wells up into the mouth from the lungs, and may stream out through the mouth or nostrils. The fluid is often stained pink. Transient albuminuria may occur.

**Differential Diagnosis** Other causes of sudden loss of consciousness must be considered, such as a cerebral vascular lesion, etc. In practice there is little difficulty in diagnosis, the physical signs in the lungs and the frothy expectoration are characteristic.

**Course and Complications** The attack may last for a few minutes or for several hours. As many as 70 attacks have been recorded in the same patient.

**Prognosis** The first attack may prove fatal, the prognosis is always extremely grave if the patient loses consciousness.

**Treatment** A hypodermic injection of morphin sulph gr  $\frac{1}{2}$  and atropin sulph gr  $\frac{1}{50}$  should be given immediately. If this fails 10 to 15 oz of blood should be removed from a vein in the arm. Lumbar puncture is usually of no avail.

### Chronic Œdema of the Lungs

This occurs in association with passive hyperæmia (see p 172) and as a manifestation of œdema in renal disease.

### Infarction of the Lungs

**Definition** Obstruction of a branch of the pulmonary artery, with resultant hæmorrhage into the lung alveoli and interstitial tissues.

**Etiology** There are two varieties, the embolic and the thrombotic.

**Pulmonary embolus** This is the most common cause. The embolus may consist of blood clot, of a fragment of an intracardiac vegetation, less frequently of air, of particles of fat, new growth, of parasites such as a hydatid daughter cyst or of droplets of amniotic fluid containing meconium. **Predisposing causes** Cardiac disease, such as mitral stenosis, venous stasis and childbirth. Surgical cases are most commonly secondary to operation in which the anterior abdominal wall has been incised. This tends to lead to stagnation in the iliac veins and their tributaries owing to inhibition of diaphragmatic respiration caused by pain in the wound with consequent venous thrombosis. Pulmonary embolus is also comparatively common in cases of fractured femur, probably due to venous stagnation, caused by the immobilisation of the limb. Fat embolus may also result from a severe shaking, multiple and compound fractures severe burns, etc. Sepsis does not appear to play an important part in the etiology of pulmonary embolus.

**Pulmonary thrombosis** This may occur in any acute or chronic lung disease, in mitral stenosis and also as a secondary process in con-

nection with a pulmonary embolus. It may also be a manifestation of phlebitis migrans (see p. 271), or occur after operations.

**Pathology.** The pulmonary embolus, when formed of blood clot, may be derived from one of the veins in the abdomen or legs. It is detached and carried through the right side of the heart to the lungs. It may also be formed from blood clot in the right auricle, in cases of congestive heart failure, or in mitral stenosis. Air embolus may result from an operation on the chest wall, as in exploring an old empyema track. Recent infarcts resemble blood clot, whereas old ones become absorbed and replaced by fibrous tissue. When the infarct extends to the lung surface, it is cone-shaped, with the base outwards, and evidence of dry pleurisy is seen on the surface. Deep-seated infarcts are oval or circular. The infarct sinks in water. It is not always possible at autopsy to discover the source of the embolus.

**Clinical Findings.** In a typical post-operative case, there is a history of an operation about 10 days previously. The patient is suddenly seized with agonising pain in the side of the chest, which is intensified with each inspiration. There may also be cough, and the immediate expectoration of blood, or of blood and frothy sputum. The pain may be so severe that the patient collapses.

**On Examination:** The patient is found sitting up in bed, much distressed, with rapid and shallow respirations. There may be no physical signs in the lungs in the early stages, beyond generalised weak air entry. In a few hours an area may be detected in which there is slight dulness, with definitely deficient breath sounds and a few fine râles. Later a pleural friction rub may appear, with signs of consolidation of a portion of the lung, usually in the lower lobe. If the infarct is basal, involving the diaphragmatic pleura, the pain is referred to the tip of the corresponding shoulder. The temperature, and pulse and respiration rates are raised. All cases do not conform to this type. There is the cardiac variety, in which the patient, after an operation, suddenly becomes pale and collapsed, suffers no pain, is dyspnoëic, and dies in a few minutes. Such cases are mistaken for heart failure until the post-mortem examination reveals the presence of a pulmonary embolus. There is also a cerebral or apoplectiform variety. Here the patient, who has been convalescing from an operation for about 2 weeks, and who perhaps is just getting out of bed, suddenly falls unconscious, is cyanosed, and has stertorous breathing. Death occurs in a few hours, and at the autopsy no cerebral hæmorrhage is found, but a pulmonary embolus is present. With fat embolism there is a symptom-free interval of a few hours to 2 days. This is followed by dyspnoea, pallor, cyanosis, restlessness and sweating. The patient is very ill and brings up frothy sputum which may be blood-tinged. Fat globules may be found in the sputum. Maternal pulmonary embolism due to amniotic fluid is said to be the commonest cause of death during labour and the following nine hours. It is associated with excessive uterine contractions and amniotic fluid containing meconium. Lesser degrees of severity may cause post-partum shock and collapse.

**Differential Diagnosis.** This is usually easy, when there is severe

pain in the chest. Hæmoptysis occurring in mitral stenosis is generally considered to be due to an infarct in the lungs, rather than to passive hyperæmia.

**Course and Complications** The signs of an infarct in the lungs usually persist for about 1 to 2 weeks, and the sputum may contain blood for about 7 to 10 days. Further infarcts may occur at short intervals. If the embolus is infected, abscess of the lung may ensue.

**Prognosis** This is very grave if the infarct is large, and in the cardiac and cerebral types of cases.

**Treatment Prophylactic** After abdominal operations, and in fractures especially of the femur, costal and diaphragmatic respiration should be as free as possible, and general muscular contractions encouraged. If venous thrombosis occurs in a limb, the affected part should be kept at rest. Sodium citrate should also be given in doses of gr 30 t.i.d.

**Curative** The immediate treatment of a pulmonary infarct is directed to the relief of pain and the alleviation of shock. Pain is most effectually abolished by strapping the affected side of the chest, from below upwards, in a position of full expiration. The strapping should include portions applied across the top of the shoulder from before backwards. A hypodermic injection should be given of morphin sulph gr 1/6, atropin, sulph gr 1/120 and strychnin hydrochlor gr 1/60. Heparin may be administered the average dose for an adult being mg 75 to 150 injected intravenously in the form of a 5% solution, 2 or 3 times a day for 4 or 5 days. In cases in which the heart has stopped beating, immediate embolectomy has been performed with some successes. The base of the pulmonary artery is opened and the clot removed.

### Hæmoptysis

**Definition** Spitting of blood.

**Etiology** Hæmoptysis may be true or spurious. In true hæmoptysis the blood is derived from the larynx, trachea, bronchi or lungs, whereas in spurious hæmoptysis the source of the bleeding is above the larynx.

The causes are very numerous. *True hæmoptysis* Pulmonary tuberculosis is the most frequent cause, next to which comes mitral stenosis, associated with pulmonary infarct, and congestive (left ventricular) failure. *Other fairly common causes are pneumonia, bronchopneumonia, infarction from any cause and bronchiectasis.* Less frequently hæmoptysis is due to a tumour of the bronchus such as an adenoma or carcinoma, abscess or gangrene of the lungs, bronchitis, spirochætal bronchitis, paragonimiasis, actinomycosis, hydatid infection, pneumoconiosis, or erosion of the lung by a pneumolith (*hæmoptysis calculosa*). Penetrating wounds of the lungs, due to a foreign body or fractured rib, are sometimes met with. Hæmoptysis may also be due to blood diseases such as purpura, leukæmia, hæmophilia, or pernicious anæmia, to deficiency diseases such as scurvy or lesser degrees of vitamin C insufficiency, to lack of



vitamins P or K; or to the hæmorrhagic forms of small-pox and measles. It is at times associated with a high blood pressure, arteriosclerosis and emphysema. There is probably no such thing as hæmoptysis due to vicarious menstruation. An aneurysm may erode into a main bronchus or into the trachea and cause a recurrent "weeping" of blood, or a fatal hæmorrhage. Other tracheal and bronchial causes include tumours, the infective *granulomata* such as tuberculosis or syphilis, and erosion of a caseous tuberculous gland.

*Spurious hæmoptysis*: The bleeding comes from the gums, from pharyngeal varices or from the nose. It is sometimes self induced and a form of malingering.

*Clinical Findings.* These vary with the amount of blood expectorated. Thus the sputum may be stained with blood, small dark clots may be expectorated, or there may be a frank hæmoptysis of bright frothy blood of several ounces, or a profuse discharge of a pint or more of blood may occur. The sputum usually contains blood for 2 or 3 days, the colour becoming darker after the bleeding has stopped, and stale blood is expectorated. Unless the bleeding is severe, the patient is unaware of it until he sees the sputum. In a sudden hæmoptysis, in which pure blood comes up, he may just "clear his throat," taste and feel a saltish warm sensation in the mouth, and then spit out the blood. Rupture of a vessel into a pulmonary cavity may cause death in a few minutes, whereas if an aneurysm bursts into a bronchus, the patient falls dead in a few seconds. In pulmonary tuberculosis hæmoptysis is more common in hot weather, and in the majority of cases it occurs when the patient is at rest. In some cases hæmoptysis leads to lobar collapse of the lungs. Deficiency of vitamins C or P may be accompanied by lowered capillary resistance (see p. 519) and by a tendency to spontaneous petechiæ and ecchymoses. Lack of vitamin K may be associated with chronic cholecystitis, jaundice and hypoprothrombinæmia and result in hæmoptysis from an old-standing tuberculous cavity.

*Differential Diagnosis.* The diagnosis of hæmoptysis presents no difficulty if the patient is seen during an attack. If there is only the history to help, there may be great difficulty in differentiating it from hæmatemesis. Attention should be paid to the following points: Was the blood coughed up or vomited? Is there a history of previous chest trouble, or of indigestion? Did the patient feel faint before the blood came up, as often is the case with hæmatemesis. Was there food mixed with the blood? Was the blood of a brownish colour, due to the action of gastric juice, or was it bright and frothy? Was there blood in the sputum for some days subsequently? Did melæna occur after the hæmorrhage? Melæna is not absolutely diagnostic of hæmatemesis, as blood may be swallowed in a severe hæmoptysis. If the blood is actually seen, the reaction to litmus should be tested. In hæmatemesis it is acid, unless the patient has taken a large dose of alkali just before the bleeding took place, whereas in hæmoptysis the reaction is alkaline. In all cases a history of a definite hæmoptysis should be treated seriously and as soon as possible further investigations should be carried out to determine its cause. In some cases, however, a cause cannot be found.

**Treatment** In cases of pulmonary tuberculosis the patient should be propped up in bed in a semi-recumbent position, and if it is known from which lung the bleeding is coming, he should be inclined slightly to that side. This helps to prevent the blood being aspirated into the healthy lung and carrying tuberculous infection there. If the bleeding is severe or if the patient cannot be calmed, a hypodermic injection of morphin sulph gr  $\frac{1}{4}$  should be given immediately. The patient must keep still and not do anything for himself. The chest may be auscultated, but percussion must not be performed. If the bleeding persists, the morphine may be repeated, but not more than a total of gr 1 should be given in 24 hours. Bandages applied to the thighs, with sufficient pressure to obstruct the venous but not the arterial circulation, sometimes help to stop the hæmorrhage. They should be kept on for half an hour, and then can be applied to the upper arms for another half hour. If the bleeding is very severe and threatening life, an endeavour should be made to collapse the affected lung by means of an artificial pneumothorax. Usually about 500 to 800 c.c. of air must be introduced into the pleural cavity to check the hæmorrhage. Cough should be allayed by a sedative linctus, such as Tinc opii camph m 20 glycerin m 20, and syr pruni serotin m 20, m 60 occasionally. Other measures which may be tried to arrest the bleeding are the inhalation of amyl nitrite m 5, the subcutaneous injection of 5 mls of Coagulen Ciba, t.i.d., the intravenous injection of 10 mls of calcium gluconate (B.P. Add.) or the daily injection of emetine hydrochloride gr 1 intramuscularly for 5 or 6 days. An intravenous injection of 10 mls of a 1% solution of congo red may also assist coagulation at the site of the bleeding. Alcohol, ergot and digitalis should not be used. A mixture is sometimes helpful containing Ol. tereb m 10, tinc quill m 10, syrup. m 30, aq cinnamon dest ad fl oz 1. Fl oz 1 t.i.d. All food should be taken cold, but it need not necessarily be fluid, thin bread and butter, jellies, cold fish and cold chicken being allowed and a saline aperient of mag sulph gr 120 should be given. Vitamin C deficiency can be diagnosed by studying the concentration of ascorbic acid in the urine before and after the administration of test doses of ascorbic acid. If there is deficiency of vitamin C ascorbic acid tab (B.P. Add.) mg 50 2 tabs t.i.d. should be given. If no response is obtained and the capillary resistance is lowered, vitamin P should be administered, as Hesperidin tab 0.25 G, 4 tabs daily. If the hæmoptysis is associated with hypoprotrombinæmia vitamin K should be prescribed, as Klotogen 1 capsule or Kapilon 1 tab t.i.d. by mouth, together with Bilein gr 5 capsule, 2 t.i.d. by mouth if there is jaundice. Alternatively, Kapilon or Prokayvit 1 to 2 mls are injected intramuscularly for 3 or 4 days.

### Abscess of the Lungs

**Definition** A localised purulent infection of the lungs

**Etiology** The abscess is caused by infection with pyogenic organisms, such as streptococci, staphylococci, pneumococci, spirochaetes or anaerobes

*Spirochaetes* are almost always present in the pus of inhalation abscesses. The abscess may be due to inhalation of some septic material, as after tonsillectomy or other operations on the naso-pharynx. In some cases inhalation of septic material from the mouth during sleep may lead to pneumonitis and pulmonary abscess. In other cases the abscess is embolic in origin, as when a tooth is extracted with a local anæsthetic, and pulmonary abscess and embolic focal nephritis subsequently develop simultaneously. Less often the abscess is secondary to bronchopneumonia or lobar pneumonia, or to an interlobar empyema, subphrenic abscess, new growth of the lungs or œsophagus, suppuration in the bronchial glands, or caries of the ribs or spine. The amoebic pulmonary abscess is described separately (see p. 688).

**Pathology.** The inhalation abscess is usually in the right lung, at any site. Embolic abscesses may be multiple, and often they are close to the pleura. The abscess may be encapsulated and closed, or in connection with a bronchus.

**Clinical Findings.** A typical example of inhalation abscess is as follows: A day or so after a guillotine operation on the tonsils, the temperature rises. After a few days the patient notices that his breath is offensive after cough. The temperature then rises higher, and the patient is acutely ill.

**On Examination:** In the early stages a small area of dulness, with diminished tactile fremitus, weak breath sounds and a few râles, is detected in one lung. Unless the abscess is in connection with a bronchus there is no purulent sputum. The temperature, and pulse and respiration rates are all raised. If the abscess ruptures into a bronchus, offensive purulent sputum is produced. This may measure about 14 oz. or more in the 24 hours. With the appearance of the pus the temperature usually falls, and the general condition of the patient is ameliorated. The blood shows a leucocytosis of about 12,000 to 15,000 per c.mm. The X-rays reveal a shadow in the lung, at first homogeneous, but after rupture into a bronchus a cavity with a fluid level may be seen when the patient is in the erect position. This may only be visible in a lateral radiogram. Lipiodol usually does not enter the affected zone.

**Differential Diagnosis.** In the early stages a pulmonary abscess may be mistaken for bronchopneumonia or an encysted interlobar empyema. After rupture has occurred, the possibility of bronchiectasis must be considered. The history of the case, and the clinical and X-ray findings usually render the diagnosis clear. It should always be remembered that a lung abscess may be associated with a bronchial carcinoma, the former being the prominent feature. The diagnosis is generally established by bronchoscopy.

**Course and Complications.** In some cases the patient coughs up the contents of the abscess, the temperature rapidly falls to normal, and a spontaneous cure is effected. In others, the expectoration is continuously purulent, and the temperature falls, with periodical rises as retention of secretion occurs. The suppurative process is slowly continuous; recurrent hæmoptysis, and eventually bronchiectasis may

result The infection may be very acute, or the patient's resistance very low, and the lung then becomes gangrenous (see p 181) Metastatic foci may form elsewhere, such as an abscess in the brain

**Prognosis** This is always grave It depends largely upon the resistance of the patient, whether the abscess is closed or in communication with a bronchus, and upon the treatment adopted

**Treatment.** If the diagnosis of pulmonary abscess has been made soon after its occurrence, a short time may be allowed to see if it will effect a spontaneous cure by rupture into a bronchus with subsequent expectoration of the pus During this period the patient should be given a course of Sulphapyridine (M & B 693) as described on p 144 A creosote solution should be inhaled from the pad of a Burney Yeo mask The following prescription may be used Creosot, sp chlorof, phenol aa 2 parts, liq iodi mit, sp ether, aa 1 part A few drops are placed on the pad of the mask every hour Postural drainage and short wave therapy should also be employed, but the latter has sometimes to be discontinued owing to hæmoptysis If a cure is not effected in 3 to 4 weeks further measures will be required An attempt should be made to empty the abscess by passing a bronchoscope under local anæsthesia and, if this is not possible, to collapse the lung by means of an artificial pneumothorax The refills should be given slowly, keeping the pressure low, and frequent X ray examinations should be made to see if the lung is collapsing, or if there are adhesions Even if the abscess is not at first in communication with a bronchus, as the lung collapses it may rupture into a bronchus, and thus be evacuated In some cases, when there are adhesions, the abscess ruptures into the pleura In such instances the patient feels a pain in the side, the temperature rises, and a pleural effusion develops A sample of fluid should at once be removed for examination Usually it is clear or slightly turbid for a day or so, and sterile It then becomes thicker and definitely offensive No delay should now be allowed for the fluid to thicken more, but a rib should at once be resected and the pleura drained In this way the abscess usually is evacuated through the pleura, and after a few weeks the temperature settles, the sputum ceases to be purulent, and the empyema wound can be allowed to close The development of a bronchial fistula may prolong convalescence The alternative method is to drain the abscess at once through the chest wall, providing the pleura over it is adherent If it is not adherent, a two stage operation is required, the first operation being concerned with rendering the pleura adherent over the abscess, and in the second operation the abscess is drained The results of this operation are in some cases good, but it may be many months before the wound in the chest wall closes, or, after healing, the wound may break down and discharge pus again Frequently the operative results are very disappointing, the patient developing a bronchial fistula and empyema Sinuses then form which have to be drained for many months Post operative fatalities from pyæmia with cerebral abscess formation are also not unknown When the abscess is complicated by bronchiectasis lobectomy may be required

## Gangrene of the Lungs

**Definition.** Putrefaction of a portion of the lungs.

**Etiology.** Gangrene of the lungs is caused by infection with pyogenic organisms and anaerobes, the resistance of the patient being very poor. *Predisposing causes:* Debility, old age, diabetes mellitus, chronic alcoholism and insanity. Gangrene of the lungs may be associated with aspiration bronchopneumonia, pulmonary embolus, especially that variety which is secondary to a bone abscess in typhoid fever, rupture of a carcinoma of the œsophagus, pressure of a thoracic aneurysm or new growth, pulmonary tuberculosis, bronchiectasis and rarely lobar pneumonia.

**Pathology.** The gangrenous area of lung is usually circumscribed, rarely diffuse. The affected zone is greenish-black, soft and sloughing, with a very offensive odour. It is surrounded by a ring of consolidated lung, outside which there is hyperæmic and œdematous lung tissue. There is also usually some generalised bronchitis.

**Clinical Findings.** A history of one of the associated causes mentioned above is usually obtained. The patient is very ill with a high fever and marked prostration. He may complain of cough and offensive sputum.

*On Examination:* If the gangrenous area is in communication with a bronchus, the breath and sputum are horribly offensive, much more so than in bronchiectasis or pulmonary abscess. The sputum is greenish-grey, or sometimes almost black, and may contain blood. On standing in a conical glass, it separates in three layers, frothy above, homogeneous in the middle, and a greenish-black deposit below. Portions of elastic tissue and lung substance may be found in the deposit on microscopical examination. The physical signs vary with the stage of necrosis, a consolidated, caseous or excavated area may be found. In some cases the pulmonary signs are obscured by those of pleural effusion. A specimen of fluid removed for examination may be slightly turbid, but teeming with organisms of various kinds, so that the pathologist returns a diagnosis of pulmonary gangrene from the fluid examination alone. There are other cases, which are not in communication with a bronchus, in which there is no offensive sputum, and in which the condition is only discovered at autopsy.

**Differential Diagnosis.** The peculiar factor of the breath and sputum, the high temperature and extreme prostration of the patient distinguish gangrene of the lung from abscess and bronchiectasis.

**Course and Complications.** The course is usually rapidly progressive. Pleural effusion, pyopneumothorax or a cerebral abscess may develop.

**Prognosis.** This is usually hopeless, although recovery has been recorded in a few cases of circumscribed gangrene.

**Treatment.** In gangrene in which there is communication with a bronchus, an attempt should be made to collapse the lung with an artificial pneumothorax. If this is not possible, owing to adhesions, surgical drainage through the chest wall, as for pulmonary abscess, may be attempted. If this is not considered feasible, an intravenous

injection of neoarsphenamine 0.3 G. should be given, and repeated in 5 days if any improvement is effected. Other measures consist in inhalation of a creosote solution from a Burney Yeo mask, as described on p. 180.

### Hydatid Disease of the Lungs

**Etiology** The disease is due to infection with the ovum of the *Echinococcus granulosus* (*Taenia echinococcus*).

**Pathology** The cyst usually forms in a lower lobe of the lungs, frequently on the right side. It is generally solitary, and surrounded by a fibrous capsule. It may rupture into a bronchus, or into the pleura, pericardium, aorta, pulmonary veins or through the diaphragm. The cyst varies in size, up to about 4 or 5 inches in diameter. In some instances its contents inspissate.

**Clinical Findings** The patient may give a history of residence in some country such as Australia, where hydatid disease is comparatively common. He may not notice any alteration in his health, and the cyst is discovered by X-ray examination. In other cases the early symptoms are those of bronchitis, or of an intra-thoracic tumour. Thus there may be cough with expectoration, or progressive dyspnoea.

**On Examination** The physical signs in the lungs may be very slight, or a definite area of dullness may be detected in one lung, with weak breath sounds and diminished voice conduction. As the cyst enlarges the mediastinal contents are displaced towards the opposite side. The blood may show an eosinophilia up to 6 or 8%, and the intradermal Casoni test (see p. 77) is usually positive. If the cyst ruptures into a bronchus hooklets may be found in the sputum.

**Differential Diagnosis** The X-ray findings are fairly characteristic, the cystic shadow having a defined margin. Other conditions, such as a dermoid cyst or a simple pulmonary tumour, may cause difficulties. The blood and cutaneous tests for hydatid are useful for confirmatory evidence.

**Course and Complications** A progressive enlargement of the cyst will give rise to increasing dyspnoea, whereas shrinking and inspissation is followed by a disappearance of all symptoms. Rupture into a bronchus may be followed by a pulmonary abscess, rupture into the pleura by shock and urticaria, and rupture into the pericardium, aorta or a pulmonary vein may cause sudden death.

**Treatment.** The cyst should not be aspirated. Thoracotomy by a skilled surgeon will often enable the cyst to be removed.

### Dermoid Cysts of the Lungs

**Clinical Findings** There are usually no symptoms until the cyst enlarges sufficiently to cause pressure effects. The patient may then complain of cough, expectoration, shortness of breath or of pain in the chest. Hairs may be seen in the sputum, and the presence of the cyst is revealed by X-ray examination. Instead of rupturing into the lung the dermoid may enlarge considerably, compressing the lung into a thin layer. The physical signs are then those of pleural effusion. On

aspiration through the chest wall the diagnosis may be established by finding hairs in the pultaceous contents of the cyst.

**Treatment.** If causing pressure effects, an attempt at removal should be made by a skilled chest surgeon.

### Congenital Cysts of the Lungs

**Etiology.** Congenital cysts are uncommon and are probably formed by a dilatation of the atria of the lungs, as no alveoli are present.

**Pathology.** In children or adults single or multiple cysts of varying size may be found, containing air or fluid. The fluid contents may be watery, mucoid or purulent. Congenital cysts are classified as: 1. The large balloon cyst which may completely compress the lung in an infant or young child. 2. The solitary cyst occupying up to half the lung field. 3. Multiple medium-sized cysts often seen near the lung root. 4. Multiple small cysts resembling bronchiectasis. They may be distinguished microscopically from bronchiectasis, for with congenital cysts the cartilage, muscle, elastic tissue and glands are irregularly distributed in the supporting tissues, the lining epithelium often remaining intact. With bronchiectasis the epithelium is destroyed, whereas the structures in the wall maintain their normal position.

**Clinical Findings.** An infant may be still-born and at autopsy the lungs are full of small cysts. In an infant or young child sudden dyspnoea may result from distension of a large cyst. Congenital cystic disease in adults may give rise to recurring attacks of dyspnoea, cyanosis and hæmoptysis. When infection occurs there is loss of weight, irregular fever, cough, offensive sputum and clubbing of the fingers. This may be followed by lung abscess, bronchiectasis, empyema or cerebral abscess. A cyst may rupture and cause a simple or benign spontaneous pneumothorax.

**Differential Diagnosis.** A single air-containing cyst may be erroneously diagnosed as a cavity due to pulmonary tuberculosis or as a pneumothorax. When infected, the clinical findings resemble those of lung abscess or bronchiectasis.

**Treatment.** This varies with the type of cyst. Pressure symptoms in a large balloon cyst must be immediately relieved by the insertion of a needle, followed later by pneumonectomy. Radical cure of unilateral infected cysts may be effected by lobectomy or pneumonectomy.

### Paragonomiasis

#### *(Pulmonary Distomiasis. Lung Fluke Disease)*

**Etiology.** The causative organism is a trematode, the *Paragonimus westermani* or *Distoma ringeri*. Infection in man occurs by drinking infected water, by bathing in it, or by eating infected crabs. The disease is endemic in Japan, Formosa, Korea, China and the Philippines.

**Pathology.** The trematode makes burrows in the lung.

**Clinical Findings.** The patient complains of recurrent hæmoptysis, often related to exercise. The diagnosis is established by finding the ova in the sputum.

**Treatment** *Prophylactic* In infected areas all drinking water must be boiled, bathing should be forbidden and crabs must not be eaten

*Curative* This is as for bilharziasis (see p 716)

## THE PLEURÆ

### Acute Dry Pleurisy

#### *(Fibrinous or Plastic Pleurisy)*

**Definition** Inflammation of the pleura, not accompanied by a fluid exudate

**Etiology** Primary cases may be due to a rheumatic infection, following exposure to cold. Pleurisy is often secondary to a tuberculous focus in the lungs, to pneumonia, pulmonary infarct, bronchiectasis, a neoplasm of the lungs, or to injury of the chest wall

**Pathology** The inflammation may be localised or diffuse, both layers of the pleura are usually involved, a sticky exudate of lymph and fibrin being found. The pleurisy may be localised to an interlobar septum, or to the diaphragmatic pleura

**Clinical Findings** In the primary cases, and in those associated with tuberculosis the patient is often a young adult who says that he was suddenly seized with a pain in the side resembling a "stitch". Any attempt at taking a deep breath or coughing intensifies the pain. It should be remembered that in some cases there may be no pain, although an extensive pleural rub is present

**On Examination** The temperature is usually a little raised 99° or 100° F. The respiration rate is also somewhat above the normal, but the breathing is shallow. The patient may prefer to lie in bed on his back or on the sound side, owing to local tenderness over the affected area

**The chest** **Inspection** The movement may be slightly diminished on the affected side. **Palpation** Often there is local tenderness at the site of the pain. **Friction fremitus** may be felt. **Percussion** The resonance is normal, or slightly diminished. **Auscultation** There is weak air entry over the area of the pleurisy. A leathery or creaking rub may be heard at the end of inspiration or beginning of expiration, or fine pleural crepitations at the end of inspiration. These are constant after cough, helping to differentiate them from pulmonary râles which often disappear after cough. Vocal resonance may be slightly diminished over the affected area

In diaphragmatic pleurisy the pain is often referred to the tip of the corresponding shoulder or to the shoulder joint (the reflex is through the phrenic nerve to the 4th cervical nerve root), or the symptoms may be abdominal. In the latter case a tender area can be found in the subcostal plane, about 2 inches from the mid line (*le bouton de Guéneau de Mussey*). There is usually weak air entry over the corresponding lower lobe of the lung. In interlobar pleurisy, the inflammation may be localised to the septum between the right upper and middle lobes. The pain is then felt in the region of the fourth right costal cartilage,



and fine crepitations may be audible at this level. Often the condition is only revealed by X-ray examination.

**Differential Diagnosis.** Other causes of pain in the chest must be considered, such as myalgia or fibrositis of the intercostal muscles or membranes (pleurodynia), intercostal neuralgia or neuritis, periostitis of a rib, the initial pain of herpes zoster, and pain referred from the heart or abdomen. The physical signs of dry pleurisy, enumerated above, are very characteristic. In intercostal myalgia, although the pain is intensified by deep breathing, no pleural signs are found. In intercostal neuritis or neuralgia the pain follows the nerve path, and localised tender spots are present, especially at the back (posterior primary division), in the mid-axilla (lateral cutaneous branch) and at the chondro-sternal junction (anterior cutaneous branch). The appearance of the typical eruption establishes the diagnosis of herpes. Rib periostitis may be shown by X-ray examination. Pain due to cardiac or gastric causes is often related to effort or to digestion.

**Course and Complications.** In simple cases the pain usually lasts for about a week, but fluid may rapidly form, and, by separating the inflamed surfaces, cause the pain to disappear. In other cases the pain of dry pleurisy is rapidly followed by the symptoms and signs of lobar pneumonia, or it may be the initial symptom of a pulmonary neoplasm or of tuberculosis. An extensive plastic pleurisy may pass on to pleurogenous fibrosis of the lungs.

**Prognosis.** This depends largely upon the cause. The possibility of pulmonary tuberculosis or of a neoplasm should always be borne in mind.

**Treatment.** The patient must be kept in bed until the temperature is normal. The pain may sometimes be relieved by the application of *liq. iodi mitis* or *Antiphlogistine* (*cataplasma kaolini B.P.*) to the affected side. If this is unsuccessful the side should be strapped from base to apex, in a position of full expiration. The useless dry cough can be checked by a sedative linctus, such as *Oxymel. scillæ m. 30*, *acid. hydrocyan. dil. m. 2*, *morphin. tartrat. gr. 1/10*, *aquam ad m. 60*. To secure sleep some drug such as aspirin *gr. 5* to *10* may be given at night. The diet during the febrile period should be fluid, and the bowels should be opened daily with salines, such as *mag. sulph. gr. 60* to *120* mane. When the temperature has fallen the chest should be X-rayed to see if there is any evidence of underlying pulmonary disease. If a tuberculous focus is discovered, the treatment is as for an early case of pulmonary tuberculosis (see p. 153). Apart from this, gentle breathing exercises should be encouraged during convalescence, to expand the lung, increase the circulation and prevent the formation of pulmonary fibrosis.

### Chronic Dry Pleurisy

Chronic thickening of the pleura may be found in association with pulmonary tuberculosis, in artificial pneumothorax or pleural effusions of some duration, after an empyema, and in polyserositis.

## Pleural Effusion

The following varieties are described 1 Clear effusions, of which there are two varieties, pleurisy with effusion (serothorax), and a pleural transudate (hydrothorax) 2 Purulent effusion, an empyema (pyothorax) 3 Hæmorrhagic effusions, such as hæmothorax, hæmoserothorax, and hæmohydrothorax 4 Milky effusions, such as chylothorax, pseudochylothorax, and chyloform effusions

## Pleurisy with Effusion

*(Sero fibrinous pleurisy Serothorax)*

**Definition** An inflammatory serous pleural exudate

**Etiology** The most important cause is a tuberculous focus in the lungs or pleura Some cases may be due to rheumatism, or be secondary to oral sepsis or may occur as a complication of septicæmia or enteric fever. Less commonly the effusion is associated with inflammatory pulmonary lesions such as pneumonia and bronchopneumonia, or with a pulmonary infarct or new growth It may also be met with as a complication of lesions of the pericardium or abdomen or as a manifestation of polyserositis Artificial pneumothorax is complicated by a serous effusion in about 50% of cases

**Pathology** A plastic pleurisy is followed by the output of a clear yellow exudate The specific gravity of the fluid is between 1.010 and 1.018 The reaction is alkaline, and protein (albumin, globulin and fibrinogen) is present to the extent of 4% or over The fluid usually clots on standing Microscopically polymorphonuclear cells, lymphocytes and a few red cells are seen, and occasionally eosinophils predominate In one case with combined abdominal tuberculosis and a tuberculous pleural effusion the fluid showed 80% and the blood 14% of eosinophils A few pleural endothelial cells may also be seen The exudate is often sterile, but tubercle bacilli may be found, or if the exudate is injected into a guinea pig it may cause the animal's death from tuberculosis Lymphocytosis is suggestive of a tuberculous infection In other cases the pneumococcus or B typhosus may be found The amount of fluid varies from a few ounces to over 2 pints

**Clinical Findings** The effusion may arise insidiously or follow an attack of acute dry pleurisy, or occur as a complication of one of the conditions mentioned above The symptoms vary with the mode of onset the patient may complain of pain or of dyspnoea The results produced by the effusion depend largely upon the amount of fluid present

**On Examination** In an average case there is fever of 101° F or over, the pulse is frequent, over 100, and the respiration rate is above normal The patient appears distressed on slight exertion, and there is some cyanosis He tends to lie on the affected side or may require to be propped up The chest Inspection There is diminished movement on the affected side The shadow of the diaphragmatic movement, as seen on the chest wall when the patient breathes, is not visible on the affected side (Litten's sign) The cardiac impulse may

be seen displaced away from the side of the effusion. Palpation : There is diminished expansion on the affected side. Tactile fremitus is usually absent over the effusion. The cardiac apex is moved away from the side of the fluid, and in a left-sided effusion cardiac pulsation may be felt to the right of the sternum. Percussion : There is stony dullness over the effusion, above the level of the fluid a high-pitched note may be elicited (skodaic resonance) due to compression of the lung. The line of the upper level of the fluid, in a moderate sized effusion, is curved from before backwards, the highest point being situated in the posterior part of the axilla (the S curve of Ellis or Damoiseau's line). The level of the upper border of the fluid may also vary with change of position of the patient. A triangular area of dullness may be found on the opposite side at the back, owing to the fluid forming a mediastinal bulge (Grocco's triangle). The apex of this triangle is on top, near the spine, at the level of the upper border of the effusion. Auscultation : The breath sounds over the fluid may be absent, very weak, or bronchial in character if the lung is collapsed and the bronchi remain patent. The air entry is usually feeble above the fluid, whereas over the opposite lung the breath sounds are loud and harsh. Some scattered rhonchi may be heard above the fluid, and a few râles at the opposite base. Usually there are no adventitious sounds over the dull area, but at times some coarse râles are audible. A pleural friction rub may be heard just above the upper fluid level. Vocal resonance is usually absent over the fluid, but near its upper level the conducted voice sound has a nasal or bleating character (ægophony). Whispering pectoriloquy and bronchophony are occasionally present over a pleural effusion. Bacelli enunciated the dictum that the whispered voice sound is conducted through a serous but not through a purulent effusion. This, however, is often not the case. A large pleural effusion may cause a downward displacement of either the liver or the spleen.

Differential Diagnosis. The nature of the fluid in the chest can only be determined by exploratory puncture. The diagnosis of the presence of a pleural effusion is often a matter of great difficulty, especially if the fluid is of small volume or loculated by pleural adhesions. The most reliable indications of fluid are the triad of sigus, stony dullness, absence of tactile fremitus, and absence of adventitious sounds. Cardiac displacement is not always present. The breath sounds are so variable that no reliance can be placed on them. A pleural effusion must be differentiated from the following conditions : Thickened pleura and fibrosis of lung, consolidation of the lung, and pericardial effusion. With a thickened pleura and fibroid lung, the dullness is not of such a stony character, the chest wall is often flattened and the heart may be displaced towards the affected side. Adventitious sounds, such as fibroid râles, are often heard over the affected lobe. In pulmonary consolidation the note also is not so stony, vocal fremitus is increased, and râles are usually audible. There is also no cardiac displacement. In pericardial effusion, the dullness has a peculiar shape (see p. 200), and the signs at the left base behind are those of consolidation (see p. 200). In all cases of doubt, especially in a localised interlobar, mediastinal or

diaphragmatic effusion, an X-ray examination is of great value, and should be obtained, if possible, before any exploratory puncture of the chest is made.

**Course and Complications.** A small pleural effusion is usually absorbed spontaneously in 2 to 3 weeks, with some resultant pleural thickening or adhesions. The compressed lung re-expands and very little abnormality can be detected subsequently. In other cases the lower portion of the lung does not completely expand, is congested, and fibrosis of the lung ensues. Acute oedema of the lungs is a rare complication during the early stage of the disease. A large effusion often will not absorb until a certain amount has been removed by aspiration. In other cases after aspiration the effusion recurs repeatedly, causing little constitutional or mechanical disturbance.

**Prognosis.** The immediate prognosis is good, but in a fair proportion of cases pulmonary tuberculosis is a sequela. Less frequently bronchiectasis ensues on fibrosis of the lung.

**Treatment.** The patient should be kept in bed as long as the temperature is raised, and, if practicable, until the effusion is absorbed. The amount of fluid ingested should be limited to 2 pints a day, and in obstinate cases a salt poor diet (see p. 455) may be given. Diuretics, such as a mixture containing Pot citrat, pot acetat, pot. tartrat.  $\bar{a}\bar{a}$  gr. 30, liq ammon acetat. m 60, aq chlorof ad fl oz 1 should be given 3 times a day, and a saline such as mag sulph gr 60 to 120 mane. The affected part of the chest should be painted every other day with liq iodi mit. A syringe full of fluid should be removed from the pleura and examined chemically, cytologically and bacteriologically. Aspiration with air replacement should be performed under the following circumstances 1. If the fluid reaches to the second rib, in the erect posture. 2 If there is marked dyspnoea or cardiac embarrassment. 3 If the fluid does not show signs of absorption in 3 weeks. 4 If there is a bilateral effusion, the larger exudate should be aspirated 5 If acute oedema of the lungs or severe collateral hyperaemia of the lungs occur. 6 If X ray examination reveals a tuberculosis focus in the lung or if tubercle bacilli are found in the sputum It is not advisable to aspirate all pleural effusions as a routine procedure. After the fluid has absorbed, the lungs should be X-rayed again, and if no evidence of tuberculosis is obtained, breathing exercises should be instituted to expand the lung and prevent the occurrence of fibrosis and of bronchiectasis. The sound side of the chest should be prevented from expanding by means of a towel held round it, or by the back of a chair pressed up against it, whilst inspiratory efforts are made for 5 minutes night and morning. If this exercise causes persistent pain or aching in the chest it should be discontinued for a time In all cases in which there is evidence of tuberculosis a course of sanatorium treatment is advisable.

### Hydrothorax

**Definition.** A transudate into the pleural space.

**Etiology.** The two main causes of hydrothorax are heart failure and renal disease. More rarely it is associated with severe anaemia,

famine œdema or deficiency of the vitamin B complex, or with thrombosis of the azygos veins, or pressure upon them by a mediastinal growth.

**Pathology.** The fluid transudes either as the result of venous stasis or owing to changes in the blood and tissues associated with renal disease. The transudate is pale yellow, the specific gravity is lower than 1.010, and a small quantity of protein (albumin and globulin) is present. This is less than 3%. It does not clot on standing. Microscopically only a few endothelial cells are present and there are no organisms. The transudate may be bilateral.

**Clinical Findings.** The history is that of the antecedent condition, usually cardiac or renal disease. An increase of dyspnœa suggests fluid in the pleura, the physical signs of which have been described on p. 186. Cardiac displacement is not generally marked. There is no disturbance of temperature, and no preliminary stage of dry pleurisy is noted. Œdema is often present elsewhere, such as in the legs, hands or back.

**Course and Complications.** The course depends largely upon the results of the treatment employed for the underlying cause.

**Prognosis.** This is grave, as in cardiac disease the transudate is an indication of circulatory failure, and in renal disease it is an expression of defective elimination.

**Treatment.** The fluid should be aspirated if it is causing distress, and the appropriate treatment given for the œdema of cardiac or renal disease, or for vitamin B deficiency.

### Empyema

#### (*Pyothorax*)

**Definition.** Pus in the pleural cavity.

**Etiology.** Empyema is most often a complication of lobar pneumonia or of bronchopneumonia. It may be syn-pneumonic or meta-pneumonic, according as to whether it develops during the acute febrile stage of pneumonia or as a complication after the temperature has fallen to normal. Other less common causes are pulmonary tuberculosis, bronchiectasis, abscess or gangrene of the lung, mediastinal abscess, carcinoma of the œsophagus, pericarditis, penetrating wounds of the chest, subphrenic abscess, septicæmia and pyæmia. Rarely it appears to develop as a primary condition. The causative organisms include the *Diplococcus pneumoniae* (pneumococcus), streptococci, the *Mycobacterium tuberculosis* (*B. tuberculosis*), the *Bacterium commune* (*B. coli*), the *Hæmophilus influenzae*, the *Bacterium friedländeri* (pneumobacillus), the *Bacterium typhosum* (*B. typhosus*), staphylococci and varieties of streptothrix. A sterile purulent effusion is met with in tuberculous pyothorax, and in some acute cases, for a day or so after the rupture of a lung abscess.

**Pathology.** The pleura becomes thickened, and the lung collapsed and fibrosed in long-standing cases. The fluid may be odourless or very offensive, it may be thin or thick, yellowish, greenish or brown. Large fibrinous flakes are often present in pneumococcal infections, whereas in a streptococcal empyema the fluid is often thin. The specific gravity

is usually over 1,030, and numerous disintegrating pus cells are present. The fluid may be free, or loculated at the base, apex, on the mediastinal surface or in an interlobar fissure. It may track through the chest wall, into the lungs or pericardium, or through the diaphragm. The distinction between empyema and pyothorax, which suggests that in the former the pus is localised by adhesions, is artificial and should be abandoned.

**Clinical Findings** Empyema is chiefly a disease of children and young adults, although it may occur at any age. In a typical metapneumonic case after an attack of lobar pneumonia, the temperature, which has fallen to normal for a few days, gradually rises again by irregular stages. The condition of the patient deteriorates, the appetite fails and shivering or sweating attacks may occur. There may be an actual rigor. As the effusion increases in size the toxæmic symptoms are more marked, the pulse and respiration rates increase and there is dyspnoea.

**On Examination** Often there are no special symptoms. The physical signs of pleural effusion described on p 186, may be found. In addition the special features met with in empyema are as follows. The patient is often pale and has a toxæmic appearance. There may be bulging of the intercostal spaces on the affected side, or œdema of the chest wall or corresponding arm. Pulsation may be seen in left-sided cases, usually near the apex of the heart (*empyema necessitatis*), when the empyema tracks through the intercostal muscles. Clubbing of the fingers may rapidly appear. The breath or sputum is offensive if the empyema is interlobar and communicates with a bronchus. There is usually a leucocytosis of between 15,000 and 20,000 per c mm. The exploratory needle reveals the presence of pus.

**Differential Diagnosis** It is easy to overlook an empyema, especially when the onset is insidious, as so often is the case in pneumonia. It should always be suspected if the temperature rises again after it has fallen to normal, unless this rise can be accounted for by some other complication such as spread of the disease, or the development of pericarditis. In all cases of doubt an exploratory puncture should be made, but in some cases the pus is thick and will only pass through a wide bore needle. An apical empyema is a source of difficulty, it may be mistaken for a pulmonary neoplasm, but in empyema the dullness does not extend across the mid sternal line, as it may do with a new growth. An interlobar empyema is to be suspected, when localised signs of fluid are discovered along the line of an interlobar septum, or if the breath is offensive on coughing. The X rays are of inestimable value in the diagnosis. The following conditions also must be excluded in certain cases. Unresolved pneumonia and fibrosis of the lung, tuberculosis or a growth of the lung, a rib abscess, an aneurysm, and a subphrenic abscess. In unresolved pneumonia and pulmonary fibrosis the heart is not displaced away from the affected side, and usually rales are heard over the dull area, and tactile fremitus is present. In doubtful cases exploratory puncture should be made. A rib abscess may be mistaken for a pointing empyema, or an aneurysm for a pulsating empyema.

The X-ray examination is invaluable in such cases. In subphrenic abscess help is obtained from the history of the antecedent condition, and the X-ray examination, which may reveal the situation of the diaphragm, as raised in a subphrenic abscess and depressed in an empyema. There is usually no lateral cardiac displacement with a subphrenic abscess. If an exploratory needle is inserted through a lower intercostal space and passed downwards until the pus is located, the needle will move with respiration in a subphrenic collection, as it must pierce the diaphragm.

**Course and Complications.** Unless the empyema is evacuated, the patient usually becomes more severely ill and will eventually die from heart failure, pyæmia, amyloid disease or cerebral abscess. In rare instances the virulence of the empyema abates and a sterile empyema results, with calcification of the pleura. Further, an empyema may develop on the opposite side, or the pus may track through the chest wall, into the mediastinum, pericardium, lung, or into the neck or abdomen. If an empyema ruptures into a bronchus it may result in spontaneous cure, or form a bronchial fistula, or cause death by suffocation. Other complications include abscess or gangrene of the lungs, bronchiectasis, pulmonary osteo-arthritis, pyæmia and abscess in skeletal muscles. Permanent pleural thickening usually remains as a sequela of an empyema.

**Prognosis.** Empyema is a serious condition, with a mortality of nearly 20%. The prognosis is naturally improved by early diagnosis and efficient treatment. The outlook is most favourable in pneumococcal infections; in streptococcal or tuberculous empyema the prognosis is more grave. In tuberculous empyema over 50% die. The danger to life is necessarily greater in bilateral empyemata.

**Treatment.** In all cases of doubt the chest should be explored. In loculated or interlobar empyema it may be necessary to perform several punctures under a general anæsthetic, the surgeon being then prepared to proceed to evacuate the pus if it is located. The usual advice is that a pneumococcal empyema should immediately be drained by rib resection, whereas in streptococcal cases, when the fluid is thin, it should be treated by repeated aspirations until the effusion is thicker and adhesions have formed which will prevent mediastinal displacement. The operation of rib resection should then be carried out. There are certain dangers in this procedure; thus undue delay may prove fatal owing to the development of pericarditis, and in an infected hæmothorax drainage should be performed immediately. In some cases of streptococcal empyema sulphanilamide 3 G. suspended in 2 to 3 oz. of sterile normal saline have been injected into the pleural cavity after aspiration of the effusion. The results, however, have not been very satisfactory. A tuberculous empyema should never be treated at first by rib resection, but the fluid should be aspirated with air replacement, and the pleural cavity washed out with methylene blue (1 in 5,000). In other cases pleurotomy with constant under-water drainage is required, followed later by phrenic avulsion and perhaps thoracoplasty. After-treatment consists in the use of suitable inspira-

tory breathing exercises to expand the lung, as described by MacMahon (*Lancet*, 1919, i 697)

### Hæmothorax

**Definition** Blood in the pleural cavity

**Etiology.** Hæmothorax may result from wounds of the chest wall or the lungs, from blast of high explosives, from fractured ribs injuring the lung, from division of pleural adhesions, or from rupture of an aneurysm

**Pathology** In gunshot wounds of the chest, the blood pours out into the pleural space and does not clot. The diaphragm is usually displaced upwards on the affected side, and the lung is collapsed. The hæmothorax may become infected with streptococci or with anaerobes. In other cases death rapidly ensues and the blood is clotted.

**Clinical Findings** The patient who has had a penetrating wound of the chest usually gives a history of hæmoptysis following directly on the wound. He then complains of pain in the chest, cough and dyspnoea.

**On Examination** The signs of a pleural effusion are found (see p 186). If the fluid is sterile, there is little or no pyrexia, but if, as is often the case, it is infected, a high temperature is registered. In addition, as air is often present in the pleural cavity together with blood (traumatic hæmopneumothorax), the signs of fluid and air may also be detected (see p 105).

**Differential Diagnosis.** The nature of the fluid is determined by exploratory puncture.

**Course and Complications** The course is usually that of an uncomplicated pleural effusion, with gradual absorption of the fluid. If the blood becomes infected the temperature suddenly rises to 102° F. or higher, and the patient becomes desperately ill. Massive collapse may develop in the opposite lung, or bronchopneumonia and generalised bronchitis.

**Prognosis** This is usually good in cases due to wounds. If the hæmothorax is due to rupture of an aneurysm, death occurs in a few minutes.

**Treatment** Aspiration of the fluid aids absorption, but this is not required in small effusions. Removal of air from the pleural cavity may be required if the pressure is high. In wounds of the chest, if the temperature rises, a specimen of the fluid should be withdrawn and examined microscopically. If the number of polymorphonuclears is excessive, a rib should immediately be resected and the fluid drained, without waiting for the results of aerobic and anaerobic cultures.

### Hæmoserotherax

**Definition** An inflammatory serous pleural exudate, containing blood.

**Etiology.** Hæmoserotherax may be met with in association with new growths of the lung, pleura or mediastinum, with pulmonary or pleural tuberculosis, or after previous aspirations of a serotherax. At times it is a complication of cirrhosis of the liver, scarlet fever, small pox, purpura or lobar pneumonia.



No special description of the clinical findings is called for, as they resemble those of sero-fibrinous pleurisy (see p. 186). The finding of blood in the pleural fluid should always be regarded as a very suspicious indication of an intra-thoracic new growth.

### Hæmohydrothorax

**Definition.** A pleural transudate containing blood.

**Etiology.** Hæmohydrothorax is most usually associated with heart failure. The clinical findings are those of hydrothorax (see p. 189).

### Chylothorax

**Definition.** A pleural effusion of chyle or of fluid containing chyle.

**Etiology.** Chylothorax results from injury of, or obstruction to, the thoracic duct. These may be due to trauma, erosion by a growth, pressure of a growth or of enlarged mediastinal glands, or to internal blockage by a parasite, such as the *Wucheria bancrofti*.

**Pathology.** The fluid is milky and contains fat. On standing, the fat forms a layer on top of the fluid.

**Clinical Findings.** No special description is called for. The nature of the fluid is discovered by thoracic puncture. The fluid should not be aspirated unless causing cardiac embarrassment or dyspnoea.

### Pseudochylothorax

**Definition.** An opalescent pleural effusion, which does not contain fat globules.

**Etiology.** Pseudochylothorax may be associated with chronic pulmonary tuberculosis, malignant disease of the lungs or pleura, and chronic disease of the heart or kidneys.

**Pathology.** The opalescence of the fluid may be caused by the presence of lecithin-globulin, calcium phosphate, cholesterin or filarise. On standing, a deposit forms, which, in the case of cholesterol effusions, is seen as delicate silvery flakes.

**Clinical Findings.** The signs are those of a pleural effusion. The nature of the fluid is determined by removing a sample. Aspiration is rarely required.

### Chyliform Effusions

**Definition.** A pleural effusion containing fat globules but no chyle.

**Etiology.** Chyliform effusions may be associated with tuberculosis or malignant disease of the lungs or pleura.

**Pathology.** The fluid has a milky appearance, and remains turbid on standing. The fat globules are probably derived from degenerating leucocytes and endothelial cells.

**Clinical Findings.** A sample of fluid should be removed for diagnostic examination.

### Pneumothorax

(including *Hydropneumothorax* and *Pyopneumothorax*)

**Definition.** Air or gas in the pleural cavity. Serous fluid (hydropneumothorax), or less often pus (pyopneumothorax), may also be present.

**Etiology.** The air or gas may be derived from various sources

- 1 The lungs Rupture of a tuberculous focus accounts for about 80% of all cases. Less frequently the pulmonary lesion, which causes pneumothorax by its rupture, is an abscess, gangrene, new growth, anthrasis, infarct or a hydatid cyst of the lungs, or the perforation is caused by rupture of an empyema into the lung, a fractured rib or an exploring needle. "Benign spontaneous pneumothorax," also called "pneumothorax in the apparently healthy," or "pneumothorax simplex" constitutes a special group of cases. It is probably due to the rupture of an emphysematous bulla or congenital cyst of the lungs.
- 2 The exterior The air may be introduced therapeutically, as in artificial pneumothorax, or accidentally, owing to a wound of the chest wall opening the pleura, or during aspiration of a pleural effusion.
- 3 The mediastinum The air may come from the œsophagus, due to rupture. This may be secondary to carcinoma, to corrosive poisons or to trauma from an œsophagoscope or bougie, or no cause may be found (see p. 21).
- 4 The abdomen Perforation of the stomach, duodenum or colon may lead to a subphrenic abscess, which tracks through the diaphragm into the pleura. A liver abscess may rupture into the lung.
- 5 The pleura Anaerobic organisms may form gas in a pleural effusion.

**Pathology** In perforation of the lung, the opening may remain patent, air passing in and out with respiration, or it may immediately seal over (closed pneumothorax), or it may form a valve, air entering the pleural cavity with each inspiration and being unable to leave it with expiration. This latter variety is known as suffocative pneumothorax. A bilateral pneumothorax may occur, due either to rupture of both lungs, or to the air escaping from one pneumothorax, through a minute opening into the opposite pleural cavity.

**Clinical Findings** A pneumothorax may occur suddenly in a patient who is apparently in good health, or as a complication of an illness which confines him to bed. This illness is usually pulmonary tuberculosis. The onset may occur while the patient is walking along the street, or be associated with a cough or some muscular effort. There is typically a sudden severe pain in the chest, often in the mid axilla, and a sensation of something snapping may be felt. The patient may immediately collapse or find great distress in breathing. The degree of distress depends upon the nature of the opening into the pleura, whether valve like or otherwise, and also upon the presence or absence of pleural adhesions, which loculate the pneumothorax and prevent mediastinal displacement. In the latter case, the onset of the pneumothorax is often symptomless, and it is only discovered on a routine examination.

**On Examination** With a diffuse pneumothorax the patient is usually propped up in bed, very distressed and short of breath. The chest. **Inspection** There is diminished movement on the affected side. The cardiac impulse may be seen displaced towards the sound side. **Palpation** Tactile fremitus is absent on the affected side. The cardiac impulse is felt displaced away from the side of the pneumothorax. **Percussion** There is a hyperresonant note over the pneumo-

thorax. If fluid is also present, the note is impaired at the base, and shifting dulness may be obtained when the chest is percussed with the patient at first erect and then reclining. The upper border of liver dulness may be obscured in right-sided cases. Auscultation: The breath sounds over the pneumothorax are weak, and may be of a metallic bronchial character. The coin-sound is often heard. This can be elicited in a modified form by flicking the chest wall over the pneumothorax with the finger nail, while listening through the stethoscope. *Harsh breath sounds are audible over the other lung.* With a shallow, left-sided pneumothorax systolic clicks, which are audible both to the patient and to the examiner, may be heard near the apex of the heart. Metallic râles or a splash (Hippocratic succussion) may be heard if fluid and air are present in the pleural space (hydropneumothorax). The pulse rate is usually about 120 and the respirations 20 to 30. The diagnosis is confirmed by removal of a small quantity of air by exploratory puncture. A small syringe is used containing a 2% solution of Novocain (procain. hydrochlor. B.P.). When the needle enters the pleural space, bubbles of air can be sucked back into the syringe. The X-ray examination will show a clear area due to the air, and a collapsed lung. If there is a hydropneumothorax the upper level of the fluid assumes a straight transverse line, when the patient is erect. The physical signs in a loculated pneumothorax may closely resemble those of a pulmonary cavity, and X-ray examination is of value in differentiating them.

**Differential Diagnosis.** There is usually no difficulty in the diagnosis of a complete spontaneous pneumothorax. A loculated pneumothorax may simulate a pulmonary cavity or an emphysematous bulla. Further, a loculated apical pneumothorax, occurring in a patient who has extensive fibroid tuberculosis of the opposite lung, may cause sudden severe dyspnoea, so that it is mistaken for asthma. A partial, left-sided pneumothorax may symptomatically closely resemble coronary disease. Careful exploratory puncture of the pleura, as described above, will demonstrate the presence of air. If a pneumothorax needle is now inserted, attached to a manometer, it can be shown that the air is in the pleural sac and not in the lung. In the former, the mean pressure is positive, whereas if the needle is in the lung, there is a respiratory excursion of pressure above and below the zero line, the mean pressure being zero. A loculated basal pneumothorax may closely simulate a perforated gastric or duodenal ulcer, but with a careful examination on the lines described above, the diagnosis can usually be made.

**Course and Complications.** The course of a spontaneous pneumothorax depends upon its cause and the nature of the opening. Recurrent attacks may occur or even bilateral pneumothorax. The occurrence of an infected pleural effusion increases the gravity of the situation.

**Prognosis.** This is often grave, death may rapidly occur from shock, or the air may gradually be absorbed. Bilateral cases are also of greater severity. In advanced cases of pulmonary tuberculosis a spontaneous pneumothorax usually ushers in the final scene, whereas

in early cases, or in "simple pneumothorax" due to rupture of an emphysematous bulla, the outlook is usually good, although recurrence occurs in about 20% of cases.

**Treatment** A hypodermic injection of morphin sulph gr  $\frac{1}{4}$  should be given immediately if the patient is in distress. If the pressure of air is causing dyspnoea and cardiac embarrassment, it must be reduced. This can best be done with the aid of a pneumothorax apparatus. The pressure of air is then easily recorded, it may be very high, sufficient to blow the fluid out of the manometer, if the tube is not controlled. Air is removed by running the fluid from one bottle to the other, in the reverse direction to that employed in inserting air into the pleural cavity, and the pressure reduced to a negative value, such as  $-2$  or  $-5$  cm water. If the needle is now kept in the pleural cavity for 5 or 10 minutes, it can be seen if the pressure is rising again. If this is occurring, it will be necessary either to remove air frequently or to keep in the pleura a needle, to which is attached a rubber tube connected by a T glass tube both with a pneumothorax apparatus to record the intrapleural pressure, and with a tube, the distal end of which dips under a disinfectant fluid, such as 1/1,000 perchloride of mercury in a bottle on the floor. A cannula with a blunt edge, and not a sharp pointed needle, should be left in the chest, as there is a risk of penetration of the lung and death from hæmoptysis. If the lung fails to expand after a month or so, an obliterative pleurisy may be induced by the injection of an irritant such as Gomenol. The dose suggested is 2 mls of a 2% solution, followed by 5 mls of a 5% solution, 10 mls of a 10% solution and 20 mls of a 20% solution, given at intervals of 5 to 7 days until fluid forms. This treatment must not be used when the pneumothorax is secondary to tuberculosis.

If a hydropneumothorax is present, aspiration of the fluid will reduce the intrapleural pressure and so relieve distress. A pyopneumothorax requires drainage by rib resection, unless it is tuberculous. In the latter case the fluid should be aspirated and the pleura washed out with methylene blue solution (1 in 5,000).

### Spontaneous Hæmopneumothorax

This is a rare condition in which air and blood escape spontaneously into the pleural cavity. In some cases the cause is a ruptured emphysematous bulla or a torn pleural adhesion, in others no source of the bleeding or air leak is discovered at autopsy. The treatment is as for spontaneous pneumothorax, the blood also being aspirated from the pleural sac. Transfusion of whole blood may be required to combat the anaemia, and if the intrapleural bleeding persists an intravenous injection should be given of 10 mls of 1% congo red solution.

### New Growths of the Pleura

Simple tumours are rare. They include a fibroma, lipoma, and angioma.

Malignant growths may be primary, such as an endothelioma, carcinoma and sarcoma, or secondary, such as a carcinoma or sarcoma.

### Endothelioma of the Pleura

**Pathology.** The growth spreads over the pleura, and may cause considerable thickening of both layers. It may also infiltrate the pericardium and heart, and extend through the diaphragm, the peritoneum being studded with minute nodules. A blood-stained pleural effusion is often present. The cells are chiefly endothelial. They may be agminated in plaques or rosettes, and show budding and mitotic changes (Foulis cells).

**Clinical Findings.** The patient is usually an adult of middle age, who complains of progressive loss of strength, with cough and shortness of breath.

**On Examination:** The patient may be wasted, and signs of a pleural effusion are present. On exploring the chest it will be noticed that the needle passes through a considerably thickened pleura. The fluid is usually blood-stained as described above. In some cases enlarged glands are found above the clavicles or in the axillæ, and subcutaneous nodules may be felt along the ribs. Air-replacement of the fluid and subsequent thoracoscopic examination are of value in establishing the diagnosis in an early case, when the area of growth can be visualised.

**Course and Complications.** The course is usually rapid. Complications include extension to other parts of the body, such as the heart and peritoneum.

**Prognosis.** Death usually occurs in 2 to 3 months from the date of diagnosis.

**Treatment.** This is usually only palliative. In some early cases surgical removal is possible. Dyspnoea can be relieved by aspiration of the fluid. Pain and cough can be treated only by sedatives, such as morphin, sulph. gr.  $\frac{1}{4}$  to  $\frac{1}{2}$ .

### Calcification of the Pleura

This may be demonstrated radiographically in certain long-standing cases of encysted empyema.

## THE MEDIASTINUM

### Mediastinitis

**Definition.** Inflammation of the mediastinal connective tissue.

**Etiology.** Acute mediastinitis is usually secondary to acute inflammation of the lungs or pericardium, or to rupture of the œsophagus or wounds of the chest wall.

**Clinical Findings.** The history and the examination of the patient usually suggest the primary cause. The patient may complain of acute pain under the sternum and in the interscapular region. If an abscess forms, it may cause mediastinal pressure (see p. 199), and it may point behind in the interscapular area.

**Treatment.** An abscess should be drained surgically.

### Mediastinal Emphysema

**Definition** Air in the mediastinal connective tissue spaces.

**Etiology** Mediastinal emphysema may result from rupture of the trachea bronchi, or œsophagus, or be secondary to acute interstitial emphysema of the lungs.

**Clinical Findings** The symptoms of mediastinal emphysema are usually masked by those of the primary cause. The patient may complain of pain under the sternum, and, on examination, a hyperresonant note is found over the sternum. The heart sounds are distant, and surgical emphysema may be detected in the neck.

**Treatment.** There is no special treatment, apart from that required for the primary condition.

### Enlarged Mediastinal Glands

**Etiology** The glands which lie in the posterior mediastinum may be enlarged from various causes such as 1 Simple inflammation in whooping cough, measles, bronchopneumonia, pneumonia, pneumoconiosis and influenza 2 Granulomatous infection, in tuberculosis and syphilis 3 Hemopoietic diseases, such as Hodgkin's disease and leukaemia 4 Malignant disease, such as sarcoma or carcinoma

### Bronchial Gland Tuberculosis

**Clinical Findings** The patient is usually a child between the ages of 5 and 10 years. He is noticed to be off colour, does not gain in weight, the appetite is poor, he is easily tired, and there is often some cough.

**On Examination** Small glands are usually palpable in the neck. The child is often pale with long eyelashes and bluish sclerotics. Dilated venules may be seen on the chest around the upper dorsal spines, and near the manubrium sterni. There is a fine downy growth of hair on the upper part of the back of the chest. The percussion note is slightly impaired over one apex at the back, the expiration is prolonged there, and an area of whispering pectoriloquy may be heard extending from the apex to the level of the 4th or 5th thoracic spine behind, and outwards into the supraspinous fossa (D Espine's sign). A ray examination will show enlarged glands if they are calcified, but those which are actively infected and not calcified will not be revealed. The evening temperature is usually raised to 99° or 100° F.

**Differential Diagnosis** Other causes of unexplained pyrexia in children must be excluded, such as rickets, rheumatism, pyelitis and worms.

**Treatment.** The child may be sent to a sanatorium for children, or treated at home or at a seaside resort, as in Thacker, on the lines laid down for the general treatment of pulmonary tuberculosis (see p. 155).

### Tumours of the Mediastinum

**Varieties** Simple tumours are rare. A lipoma may fill the anterior mediastinum. Other simple tumours include a myoma, chondroma, osteochondroma, persistent thymus and retrosternal goitre.

*Malignant tumours* may be carcinoma, sarcoma or ganglio-neuroblastoma. Carcinoma probably is always secondary to a growth in the bronchi or lungs, and some of the mediastinal sarcomata formerly described are probably of the nature of oat-celled carcinoma of the bronchus. Lymphosarcoma, Hodgkin's disease or leukaemia may also affect the mediastinal glands.

**Clinical Findings.** The special features of new growth of the mediastinum are the pressure effects, which are known as *the mediastinal syndrome*. Certain structures may be compressed. The trachea or bronchi: There is cough, dyspnoea or stridor and collapse of the lung. The nerves: The vagus, recurrent laryngeal, sympathetic, phrenic or intercostals may be affected. Pressure may result in slowing or acceleration of the pulse, hoarseness, inequality of the pupils, hiccough, paradoxical movement of one side of the diaphragm (up with inspiration and down with expiration), and pain in the chest or arm. The arteries: The pulses may be unequal, or the blood supply to one lung may be interfered with, and gangrene ensue. The veins: The superficial veins of the chest may dilate (see Fig. 48). The superior vena cava may be obstructed, with reversal of flow in the veins in the chest wall, the current being now from above downwards in the upper part of the chest. Oedema may also be seen in the face, neck, chest or arm, the lungs may be hyperaemic from back pressure, or a pleural effusion may develop. The lungs: A portion of the lungs may collapse. The oesophagus: Pressure will result in dysphagia. The thoracic duct: A chylous pleural effusion may develop. The early symptoms are thus very variable; a persistent ineffective cough may be complained of, or pain in the chest or arm may be first noted. The symptoms and signs are usually indistinguishable from those of new growth of the lungs (see p. 163). The diagnosis is aided by the X-ray examination, and the determination of the Wassermann reaction, which aids in the exclusion of an aneurysm.

**Treatment.** This is purely palliative in all malignant cases, as the prognosis is hopeless. A simple tumour, such as a lipoma of the anterior mediastinum or retrosternal goitre, can frequently be removed surgically. X-ray treatment should be given in cases of Hodgkin's disease or leukaemia.

### Cysts of the Mediastinum

A dermoid or a hydatid cyst is occasionally met with (see also p. 182). Treatment is surgical.

## THE DIAPHRAGM

### Spasm of the Diaphragm

**Varieties.** The spasm may be clonic or tonic.

**Etiology.** *Clonic spasm* (*singultus* or hiccough). This may be due to alimentary, nervous or renal causes. 1. Alimentary causes include: Irritation of the oesophagus or stomach, as by tobacco smoke and pungent articles of food, it may also occur in association with dilatation

of the stomach, gastritis, enteritis, peritonitis or intestinal obstruction

2 Nervous causes may be central, such as a cerebral tumour, meningitis, epilepsy, encephalitis lethargica or hysteria, or peripheral, such as a reflex from a mediastinal tumour, pericardial effusion or diaphragmatic pleurisy

3 Renal causes, as in chronic nephritis or uræmia

*Tonic spasm* of the diaphragm may be due to strychnine poisoning, tetanus, hydrophobia or laryngismus stridulus

**Treatment** If possible the cause should be removed. The clonic spasm may sometimes be abolished by holding the breath, or by drinking a glass of water. In severe cases it may be necessary to prescribe sedatives such as Chlorotone (chlorbutol B.P.) gr 5 in a cachet t d s, Luminal (phenobarbitonum B.P.) gr  $\frac{1}{2}$  to 1 t d s, or the hypodermic injection of diamorphin hydrochlor gr  $\frac{1}{4}$ , the inhalation of CO<sub>2</sub> by breathing in and out of a paper bag held tightly over the nose and mouth, or even the administration of chloroform

### Diaphragmatic Pleurisy

This has been referred to on p 184

### Diaphragmatic Paralysis

**Etiology** This may be due to central causes affecting the phrenic nuclei such as poliomyelitis, a hæmorrhage or a tumour. The phrenic nerve may be involved by neuritis or by pressure of a tumour, or it may be crushed or divided surgically or after division avulsed from its diaphragmatic terminations. It is possible that in some cases the paralysis occurs reflexly (see p 169, Massive Collapse)

**Clinical Findings** X-ray examination will show the extent of diaphragmatic movement. One half may be completely paralysed, in which case the diaphragm on that side is usually raised, or it may show paradoxical movement on the affected side, rising with inspiration and falling with expiration. This is often associated with a mediastinal neoplasm

### Hernia of the Diaphragm

#### (Thoracic Stomach)

**Definition** Protrusion of an abdominal viscus, usually the stomach, through the diaphragm into the thoracic cavity

**Etiology** This may be a congenital maldevelopment, such as a short œsophagus, or result from degeneration of the œsophageal ring of the diaphragm or from a muscular strain causing rupture, or it may be caused by a wound involving the diaphragm. Three types are described

- 1 Congenital short œsophagus with partial or complete thoracic stomach
- 2 Œsophageal hiatus (para œsophageal) hernia. The œsophagus is of normal length and does not form part of the hernia. This is the most common variety
- 3 Œsophageal hiatus hernia with shortened œsophagus, the lower end of which forms part of the hernial contents

**Clinical Findings.** There may be no symptoms or epigastric pain with distention and heartburn may be noted. In some cases the



symptoms are cardio-respiratory, resembling angina pectoris. The stomach may become nipped in the diaphragmatic opening, giving rise to severe vomiting. There may also be hiccough, dyspnoea and loss of weight, or severe cough when the patient lies on the back or side.

*On Examination:* The patient is usually an adult of over 50 years. The heart may be displaced upwards and away from the affected side. A hyperresonant note is found over the lower part of the chest in the axilla, which may simulate a pneumothorax. The diagnosis is established by a barium swallow and meal, with the patient in the Trendelenberg position.

*Treatment.* Medical treatment consists of small, well-masticated meals taken at more frequent intervals than usual. Alkalis are given for heartburn. If symptoms persist, surgery should be considered. The para-oesophageal type is the most suitable for surgical repair of the hernia.

### Eventration of the Diaphragm

*Definition.* A bulging of the affected side of the diaphragm into the thorax.

*Etiology.* The eventration is probably due to a congenital weakness of the diaphragm, rather than to an affection of the phrenic nerve; but in some cases there may be a birth injury of the phrenic nerve.

*Pathology.* The affected half of the diaphragm is thin, translucent, and consists of fibrous tissue with a few muscle fibres. The phrenic nerve on the affected side may be smaller than its fellow, but does not show evidence of neuritis. In about 94% of cases the left side of the diaphragm is affected.

*Clinical Findings.* The patient does not usually make any complaint pointing to a lesion of the diaphragm, but the signs found on examination may resemble those of a diaphragmatic hernia. The lesion is diagnosed by X-ray examination, the affected side of the diaphragm may lie at the level of the third costal cartilage, and show paradoxical movement.

*Treatment.* No special treatment is required.

### Paroxysmal Flutter of the Diaphragm

This is a rare condition in which the symptoms resemble those of angina pectoris. Screen examination shows rapid oscillations of one-half of the diaphragm, superimposed on somewhat jerky movements synchronous with thoracic respiration. Rapid relief has been recorded by spraying the skin of the neck over the phrenic nerve with ethyl chloride.

## CHAPTER III

### THE CARDIO-VASCULAR SYSTEM

**Introductory** In this section attention will be directed to the pericardium, heart, arteries and veins. The indications for special investigations will be noted. These include X ray examinations to determine the size, shape, position and movements of the heart and aorta, electrocardiograms and blood pressure readings.

#### The X-ray Appearances of the Heart

The size and outline of the heart shadow can be determined either by a teloradiogram or by an orthodiagram. In the former an X ray photograph is taken with the tube at a distance of 2 metres (6 feet) from the patient, so that there is little distortion of the heart shadow. With the orthodiagram a small beam of rays is moved round the outline of the heart, and its edge can thus be delineated with a pencil on the

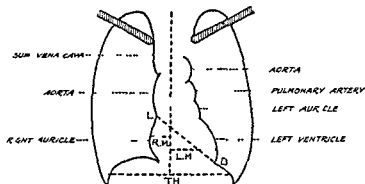


FIG 10. DIAGRAM OF NORMAL ORTHODIAGRAM—A-P VIEW

**Average Measurements** LD = Long diameter = 13.2 cm RM = Rt. medial = 4.2 cm LM = Lt. medial = 8.5 cm TD = Transverse diameter = RM + LM TH = Internal diameter of thorax Cardio thoracic ratio =  $\frac{TD}{TH} = \frac{1}{2}$

screen or on paper. The outline is drawn in expiration and during the diastolic phase of cardiac activity. A diagram showing the main features of a normal heart shadow is given (see Fig 10). X ray examinations in the antero posterior and oblique positions will show alterations in the size and shape of the heart, of its chambers and of the aorta. It will also reveal calcification of the heart, aorta or pericardium, and erosion of ribs by the hypertrophied intercostal arteries, in some cases of coarctation of the aorta.

**X-ray Kymography** This provides a record on a film of the movements of the heart and aorta. A grid, made of a lead plate, with narrow

horizontal slits equidistant from each other, is placed between the patient and the film. The grid moves slowly downwards during the exposure, which lasts 3 seconds. The heart outline appears on the film with a serrated edge, each serration corresponding with a beat of the heart. Diminution or absence of pulsations of a part of the heart, as may occur with a cardiac aneurysm, results in diminution or absence of serrations over the corresponding portion of the heart outline. Kymography appears of value chiefly in the diagnosis of a cardiac aneurysm or pericardial effusion, or in differentiating a pulsating mediastinal tumour from an aortic aneurysm. Some authorities, however, consider that no more information is given by this method than can be obtained from a screen and film examination of the patient.

**The Normal Electrocardiogram.** This is illustrated in Fig. 11. The P wave is due to auricular systole and constitutes the auricular complex.

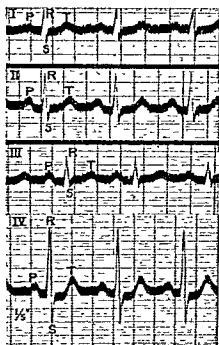


FIG. 11. NORMAL ELECTROCARDIOGRAM (Lead IVR, left arm electrode anteriorly, right arm electrode on right arm).

QRST occur during ventricular systole and constitute the ventricular complex. Q and S are often absent. T to P represents diastole.

## THE PERICARDIUM

### Acute Fibrinous Pericarditis

#### (Dry Pericarditis. Plastic Pericarditis)

**Definition.** Acute inflammation of the pericardium, with little or no fluid formation.

**Etiology** *Varities* 1 Primary cases, so-called, are rare, and may be associated with an undiscovered septic focus. In some instances, especially in children, they are tuberculous or pneumococcal. Traumatic pericarditis results from a wound or a foreign body penetrating the chest wall or the œsophagus and rarely from a non-penetrating chest injury (see p 230). 2 Secondary cases. These may be due to 1 Infections especially rheumatic fever and less often chorea. Other causes include focal infections in various sites such as the mouth and cranial sinuses, pneumonia, septicæmia as in purpural fever and osteomyelitis, pyæmia and tuberculosis. 2 Specific fevers, such as scarlet fever, diphtheria, measles, small pox and enteric fever. 3 Local extension of disease, as from inflammation or a new growth of the chest wall, mediastinum, pleura, lungs or abdomen. An infarct of the heart is often associated with pericarditis. 4 Other diseases such as nephritis, diabetes mellitus, leukæmia and scurvy in which the pericarditis is a terminal event. The exciting organisms include the *Diplococcus pneumoniae* (pneumococcus), the streptococcus, the staphylococcus, the *Neisseria gonorrhœa* (gonococcus), the *Bacterium commune* (*B. coli*), the *Bacterium typhosum* (*B. typhosus*), the *Mycobacterium tuberculosis* (*B. tuberculosis*) and possibly the *diplococcus rheumaticus*.

**Pathology** A fibrinous exudate forms, which is localised or diffuse. There may be a shaggy layer, giving a "bread and butter" effect. A small serous exudate is not uncommon. Evidence of an old healed lesion is sometimes found at autopsy in the form of a thickened white area or "milk spot."

**Clinical Findings** If the patient is a child, he is frequently suffering from rheumatism or chorea; dry pericarditis in an adult may be due to any of the causes mentioned above, whereas in old age it is often a terminal event. Pain is complained of, usually localised to the præcordium, but at times radiating to the neck, arm or abdomen. It is generally intensified by breathing, coughing or moving. In some cases only slight discomfort or a dull ache is noticed.

**On Examination** The patient usually looks ill and anxious, and he may be pale. The temperature is raised to about  $101^{\circ} F$ , and the pulse rate to 100 or 120. The chest. *Inspection* A rapidly beating cardiac impulse is often seen. *Palpation* A "to and fro" pericardial friction rub may be felt. *Percussion* The cardiac dulness is sometimes, but not always enlarged, but such enlargement is not due to the pericarditis. *Auscultation* The diagnostic friction rub is heard. This is a "to and fro" rub, not quite synchronous with systole and diastole, and usually intensified by pressure of the stethoscope. It may be very faint, or loud and creaking (*bruit de cuir neuf*) and audible without a stethoscope. It may be diffuse, or localised to the base or apex of the heart. In some cases it is heard better on deep inspiration, or when the patient is sitting. Further, it is sometimes inconstant, being heard at one examination, and not at the next.

**Differential Diagnosis** The pain must be distinguished from that due to pleurisy or to angina pectoris. Dry pericarditis is often a complication of coronary obstruction. The rub has to be differentiated

from pleuro-pericardial or pleural friction, and from a double aortic murmur. With pleuro-pericardial friction the rub is generally heard at the left or right border of the heart, disappearing with full inspiration, and returning with expiration. With pleural friction the rub may disappear completely when the breath is held. With a double aortic murmur the characteristic features are the site and area of conduction of the murmur, the absence of pain, the blood pressure readings and the cardiac enlargement (see p. 249).

**Course and Complications.** In the majority of cases the pericarditis resolves, without the formation of an effusion which can be detected clinically. In some instances a serous, hæmorrhagic or purulent pericardial exudate develops. Adherent pericardium is a likely sequela.

**Prognosis.** This must vary with the cause, but pericarditis is always a serious condition, and if death does not ensue, time alone will show what permanent damage will remain. In chronic nephritis, pericarditis is usually a signal of impending death.

**Treatment.** The patient must be kept strictly at rest in bed, lying nearly flat, or slightly propped up, or turned a little to one side or the other. Gaultherium ointment (ung. methyl. salicyl. B.P.C.) on lint should be applied daily to the præcordium. Pain is relieved by the injection of morphin. sulph. gr.  $\frac{1}{6}$  to  $\frac{1}{4}$ , or by pulv. ipecac. et opii gr. 10, for an adult, at night. In rheumatic cases a mixture containing Sod. salicyl. gr. 10, sod. bicarb. gr. 20, syr. aurant. m. 30, aquam ad fl. oz. 1 is given, fl. oz. 1 t.d.s. p.c. A careful watch should be kept as signs of collapse may occur. Should this happen, the salicylate mixture is discontinued, and a stimulant one is ordered, containing Sp. ammon. aromat. m. 20, tnc. nuc. vom. m. 7, tnc. digital. m. 5, sp. chlorof. m. 7, aquam ad fl. oz. 1. Fl. oz. 1 t.d.s. p.c. The diet should be fluid or semi-solid during the acute stage. The patient must be kept in bed for at least 3 months after the temperature and pulse have returned to normal, without salicylates being taken. Subsequently very limited walking exercise is allowed for 9 months, and no games should be played for 2 years.

### Pericardial Effusion

The following varieties may occur: 1. Clear: Sero-fibrinous pericarditis. Hydropericardium. 2. Purulent: Pyopericardium. 3. Hæmorrhagic: Hæmopericardium. Hæmoseropericardium. Hæmohydropericardium.

### Pericarditis with Effusion

(*Sero-fibrinous Pericarditis. Seropericardium. Acute Constrictive Pericarditis*)

**Definition.** An inflammatory serous pericardial exudate.

**Etiology.** Pericarditis with effusion is usually preceded by acute fibrinous pericarditis, but in some cases, especially in children, it develops insidiously.

**Pathology.** The fluid varies in amount from a few ounces to 2 or 3 pints. It is yellowish-green in colour, clear or slightly turbid, specific

gravity about 1,018, and it clots on standing. The pericardium is thickened by a fibrinous exudate and there is usually myocardial degeneration.

**Clinical Findings** The patient is a child or an adult. Usually there is a history of acute dry pericarditis, in the course of which the effusion occurred. Rarely the pericardial effusion is discovered without such a history, the patient being a child, who is off colour, looks pale and complains of shortness of breath. The patient with such an effusion complains chiefly of dyspnoea, a feeling of distress or actual pain in the præcordium and palpitations. Increasing mediastinal pressure may result in dysphagia, vomiting, hiccough, cough or hoarseness due to recurrent laryngeal paralysis. Insomnia is often a very troublesome feature.

**On Examination** The patient is pale and the lips and ears may be somewhat cyanosed. He is often restless and cannot lie flat on his back, wishing to be propped up or turned on the left side. The cervical veins may be distended. The chest. **Inspection** There may be bulging of the præcordium, with diminished expansion on the left side of the chest. Often no cardiac impulse is visible, but a diffuse wave may be seen over the third, fourth and fifth left intercostal spaces. Diaphragmatic movement may be abolished or weakened on the left side. **Palpation** The cardiac impulse is feeble or absent. Pericardial friction if previously present, disappears. Oedema of the chest-wall over the præcordium may be demonstrated by pitting on pressure. **Percussion** The area and intensity of the cardiac dulness are increased. It is described as pear shaped, with the stalk upwards. The upper level of dulness may alter with the patient in the erect and recumbent positions. The dulness obliterates the normal area of pulmonary resonance in the cardio-phrenic angle at the fifth right space, near the sternum (Roth's sign). On the left side the dulness may extend out to the axilla. **Auscultation** The heart sounds become progressively weaker as the effusion increases. A friction rub, if previously present, disappears, although it may remain at the base, or be audible in the erect but not in the recumbent position. The apex of the heart, as judged by the intensity of the heart sounds, lies internal to and usually above the apex of the cardiac dulness. The lungs. The pressure of the pericardial effusion, especially if localised to the posterior part of the pericardial sac, may give rise to special signs. Thus a small area of dulness may be found near the inferior angle of the left scapula with bronchial breathing, bronchophony and whispering pectoriloquy (Bamberger's or Lwart's sign). The pulse is usually rapid, and it may increase in force with expiration and weaken with inspiration (the pulsus paradoxus). The blood usually shows a leucocytosis. An X-ray examination will generally reveal a characteristic shadow of the effusion. This is globular in the recumbent position and pyriform when the patient is erect. With a dilated heart the shadow remains practically unaltered in shape with change of posture. The absence of normal pulsations is shown by X-ray kymography.

**Differential Diagnosis** A pericardial effusion may be mistaken for

a dilated heart or for a left-sided pleural effusion. In dilatation of the heart the apex beat is usually palpable, although weak. The dulness over the heart is not so stony in character, and Rotch's sign (see p. 206) is not present. The shape of the dulness is also different, the upper border being more flat and at a lower level. The heart sounds are more distinctly heard, pressure signs are not found behind at the left base, and the X-ray shadow differs, as described above. With a left-sided pleural effusion the heart is displaced towards the right, the heart sounds are clear, and, unless the fluid is loculated, the signs of fluid are found at the left base behind (see p. 186). X-ray examination will also serve to differentiate.

**Course and Complications.** With recovery the fluid absorbs and adhesions remain between the layers of the pericardium. The rate of absorption varies, from a few days to several weeks. Reappearance of the friction rub is an indication that this has taken place, and pain may or may not recur. In some instances the fluid becomes purulent.

**Prognosis.** A pericardial effusion is always a serious event, the immediate prognosis varying with the underlying cause. Thus with the infectious fevers recovery usually occurs, but in severe septic infections a pericardial effusion is generally the herald of death.

**Treatment.** The general treatment is as for acute fibrinous pericarditis (see p. 205). Special treatment involves paracentesis of the pericardium. This should not be lightly undertaken, as the results are very discouraging. It may be necessary if the effusion increases and seriously embarrasses the heart's action, as shown by increasing rapidity and weakness of the pulse, greater dyspnoea and restlessness, or if pus is suspected (see p. 208). Novocain (procain. hydrochlor. B.P.), 1 mil. of a 2% solution, should be injected in the fourth left space close to the edge of the sternum, or 1 inch external to it, in order to avoid the internal mammary artery, until the pericardium is pierced, when the fluid can be withdrawn into the syringe. A fine trocar and cannula is then inserted, and the fluid allowed to drain away. If, however, pericardial friction is audible at this site, the puncture should be made either in the fifth or sixth left space just internal to the left border of the pericardial dulness, or in the angle between the ensiform cartilage and the left costal cartilages, the needle then being passed upwards and backwards until the pericardium is pierced. A loculated posterior pericardial effusion may be aspirated from the back. The left arm is brought forward and the needle is inserted in the seventh or eighth left space in the mid-scapular line, to a depth of 2 or 3 inches.

### Hydropericardium

**Definition.** A transudate of fluid into the pericardium.

**Etiology.** Hydropericardium is most frequently associated with heart failure or chronic nephritis. Rarely it is due to mediastinal venous obstruction from enlarged glands, new growths or aneurysm, or to the severe cachexia of leukaemia, pernicious anaemia, or of beri-beri.

**Pathology.** The fluid is clear, pale, of low specific gravity (about 1.012), and contains less than 3% of protein.

**Clinical Findings** The symptoms are usually masked by those of the primary condition. A considerable amount of fluid may accumulate without definite symptoms as there is no preliminary stage of pericardial friction and the rate of output of the transudate is slow. The physical signs correspond with those described for sero fibrinous pericarditis (see p 206). There is usually œdema in other parts of the body.

**Treatment** It is rarely necessary to tap the pericardium and the treatment is that appropriate to the causative condition.

### Purulent Pericarditis

#### (Pyopericardium)

**Definition** Pus in the pericardium.

**Etiology** Purulent pericarditis is usually a complication of pyæmia especially when there is osteomyelitis, or it is associated with tuberculosis or new growths of the pericardium. An empyema or pulmonary abscess may lead to direct spread of infection to the pericardium.

**Clinical Findings** In addition to the symptoms and signs of sero fibrinous pericarditis the temperature shows marked diurnal variations the pulse is more rapid and sweats or rigors may occur. There is generally a leucocytosis of over 12 000 per c mm. In rare cases the patient is afebrile.

**Differential Diagnosis** The nature of the fluid can only be determined by exploratory puncture.

**Course and Complications** The disease is usually rapidly fatal but the course may be very prolonged in tuberculous infections and there may be calcification of the pericardium. The fluid may rupture through to the left pleura with empyema formation.

**Prognosis** This is very grave. Some cases of tuberculous pyopericardium undergo a spontaneous cure.

**Treatment** The pericardial sac should be aspirated the needle being inserted between the ensiform cartilage and the left costal cartilages in order to avoid infecting the pleura. If the patient's general condition permits and the fluid is not tuberculous the pericardium should be drained surgically.

### Hæmopericardium

**Definition** Blood in the pericardium.

**Etiology** The blood enters the pericardium from rupture of the heart, coronary artery or intrapericardial portion of the aorta. The cardiac leak may result from an infarct, an aneurysm of the heart, or it may be due to external trauma or to a perforating œsophageal wound. Hæmopericardium is a rare occurrence in purpura or scurvy.

**Clinical Findings** The patient suddenly dies.

### Hæmoseropericardium

**Definition.** An inflammatory serous pericardial exudate, containing blood.



**Etiology.** Hæmoseropericardium is usually due to malignant disease or tuberculosis of the pericardium.

**Clinical Findings.** These closely resemble the findings in sero-fibrinous pericarditis.

### Hæmohydropericardium

**Definition.** A pericardial transudate, containing blood.

**Etiology.** Hæmohydropericardium is usually associated with heart failure, or with malignant disease of the pericardium.

### Calcified Pericardium

This may be a sequela of acute inflammatory pericarditis, especially pyopericardium, or of a chronic pericardial effusion. It is usually discovered by X-ray examination. It may cause no symptoms, or result in cardiac distress.

### Pyopneumopericardium

**Definition.** Gas and pus in the pericardium.

**Etiology.** The gas may come from various sources, such as:

1. The exterior: In trauma from wounds or by operation on the pericardium.
2. The lungs: In tuberculosis, gangrene or pyopneumothorax.
3. The abdomen: In subphrenic abscess.
4. The œsophagus: With an ulcerating growth.
5. Gas-producing organisms, in a pericardial effusion. This is generally a post-mortem event.

**Pathology.** A purulent exudate is present.

**Clinical Findings.** The patient may complain of sudden præcordial pain, but at times the onset is insidious.

**On Examination:** Typical signs depend upon the presence of gas and fluid in the pericardium. There is an area of hyperresonance over the præcordium, which may move with alteration in the position of the patient. On auscultation a loud churning sound is heard.

**Differential Diagnosis.** If gas alone is present, it may be loculated behind the heart and cause no definite signs. When in front of the heart the hyperresonance may suggest acute interstitial emphysema which has spread to the anterior mediastinum. An X-ray examination will establish the diagnosis, the outline of the pericardium standing out as a definite line, with a translucent space between it and the heart shadow.

**Treatment.** This is similar to that described for acute sero-fibrinous pericarditis. The air may be allowed to escape through a needle if there is severe cardiac distress.

### Adherent Pericardium

#### (Chronic Mediastino-pericarditis)

**Definition.** Adhesions between the layers of the pericardium, or between the pericardium and surrounding structures.

**Etiology.** Adherent pericardium is usually a sequel of acute pericarditis. The majority of cases are therefore rheumatic, the minority being caused by tuberculosis, malignant disease and polysclerosis.

**Pathology** There are three anatomical varieties 1 Adhesions are present between the visceral and parietal pericardial layers 2 The adhesions also extend outwards to the diaphragm, chest wall and pleura (*pericarditis interna et externa*) 3 There is also thickening of the mediastinal connective tissue (chronic indurative mediastino pericarditis) Adherent pericardium is also classified as constrictive and adhesive or non constrictive, according to whether or not there is interference with the filling and emptying of the heart The chief obstruction is usually to the diastolic filling, inflow stasis resulting The heart is often not enlarged unless valvular disease is also present Calcification of the pericardium may occur

**Clinical Findings** If the adhesions are limited to the two pericardial layers, there are usually no special symptoms or signs With external adhesions the results depend upon the extent to which the heart is anchored to the chest wall and surrounding structures, and to the obstruction to the venæ cavæ and hepatic veins from mediastinal fibrosis The congestion is due to the mechanical effect of the adhesions and not to myocardial failure In a typical case of pericarditis with external adhesions the patient is often a child or young adult A history of rheumatic fever can usually be obtained The patient may complain of shortness of breath on exertion, of palpitations and præcordial distress

**On Examination** **Inspection** There is usually pallor, and the lips may be a little cyanosed The præcordial area may be prominent and a diffuse area of cardiac pulsation is seen, with a wavy impulse in the fourth, fifth and sixth left intercostal spaces The veins in the neck may be engorged and undergo some collapse with each diastole (Liedreich's sign) Systolic retraction may be noted around the apex (periapical retraction) or extensively over the præcordium Systolic retraction may also be seen in the tenth or eleventh space behind, on the left side in the scapular angle line (Broadbent's sign) **Palpation** The cardiac impulse is forcible and the apex beat is fixed, not moving outwards when the patient turns on his left side A diastolic or rebound shock is not often felt **Percussion** The area of cardiac dulness is increased downwards and to the left and often also to the right The upper border of cardiac dulness may not be encroached upon by the pulmonary resonance when the patient takes a deep breath (C D J Williams' sign) **Auscultation** In some cases a presystolic murmur may be heard at the apex, not due to mitral stenosis Often an apical systolic murmur is heard The pulse This may weaken during inspiration and become stronger with expiration (*pulsus paradoxus*) In indurative mediastino-pericarditis, fibrosis occurs in the posterior mediastinum and there may be obstruction to the superior or inferior vena cava The patient may have a hepatic aspect, there being enlargement of the liver (*pseudo-cirrhosis*) and ascites, with perihepatitis (*frosted liver*) This constitutes Pick's disease and is probably non rheumatic in origin

**Differential Diagnosis** Adherent pericardium is frequently overlooked, but the most reliable signs are the cardiac hypertrophy, fixation of the apex, and systolic retraction

**Course and Complications.** The course is usually slowly progressive in rheumatic cases. In malignant disease death is due to the growth rather than to the pericardial adhesions. Complications result from circulatory failure.

**Prognosis.** This is unfavourable, and patients with external adhesions usually die young.

**Treatment.** There is no medical cure once the adhesions have developed. It is possible that adequate rest after acute pericarditis serves to prevent their formation. The patient must live within the reserves of his cardiac efficiency. The operation of pericardial resection, with removal of the bands which constrict the venæ cavæ, offers the only hope of cure. Some wonderfully successful results have been obtained.

### New Growths and Cysts of the Pericardium

The pericardium may be invaded by an endothelioma of the pleura, by spread of carcinoma or by a primary sarcoma. A hydatid cyst sometimes develops in the pericardium and a dermoid cyst of the mediastinum may rupture into it.

## THE NEURO-MYOCARDIUM

### Disorders of Rate and Rhythm

**Introductory.** Normally the impulses for the heart beat arise in the sino-auricular node in the right auricle, whence waves of excitation spread over both auricles to the auriculo-ventricular node, situated at the posterior and right edge of the interauricular septum. The impulse then spreads to the ventricles along the bundle of His. The S-A node is under a double nervous control, the sympathetic accelerating and the vagus retarding the output of stimuli. Normal cardiac activity is therefore a sinus rhythm. Cardiac irregularities may be due to:

1. Sinus disorders: Simple tachycardia. Simple bradycardia and sinus arrhythmia.
2. Increased muscular irritability: Paroxysmal tachycardia. Premature systoles. Auricular fibrillation. Auricular flutter. Ventricular fibrillation.
3. Diminished conductivity: Heart-block.
4. Defective contractibility: Pulsus alternans.
5. Disturbed diastolic filling: Pulsus paradoxus.

These varieties will now be briefly considered.

### Simple Tachycardia

**Etiology.** Simple tachycardia may result from diverse causes, such as exercise, emotion, fevers, hyperthyroidism, chronic infections, anæmia, hæmorrhage, alcohol, cordite, atropine, tea, coffee, tobacco and thyroid extract.

**Clinical Findings.** The pulse rate at rest is usually between 90 and 120, and after exercise it may rise to 150 or 180, taking longer than 2 minutes to return to its resting rate. In such a case the exercise tolerance is considered fair, moderate or poor, according to the pulse figures and the distress engendered by the test. In other instances there are attacks of palpitations, during which the patient is conscious of the heart's action. Throbbing may be felt in the neck, and there may be

giddiness or buzzing in the ears. A sense of præcordial distress or pain may also be noticed.

*On Examination* There is usually no cardiac enlargement. A soft systolic murmur heard at the apex or base may accompany the first sound.

*Treatment* Digitalis is not usually of value unless an organic cardiac lesion is present. Bromide in the form of ammonium bromide 5 to 10 tds is often helpful. A search should always be made for an underlying cause, such as a septic focus, pulmonary tuberculosis or hyperthyroidism.

### The Effort Syndrome

(*Da Costa's Syndrome* *Disorderly Action of the Heart* D A H  
*Soldier's Heart* *Neuro circulatory Asthenia*)

*Etiology* This syndrome is a frequent occurrence in wartime amongst untrainable or imperfectly trained soldiers. In civilian life it is said to be more common in women. Various views are held as to its nature. 1 That it is a neurosis. 2 That it is due to chronic sepsis. 3 That it results from over smoking. 4 That it is the product of effort and poor physique. It is unlikely that effort alone will cause it, and the symptoms do not resemble those produced by strenuous exercise in a healthy person. In the majority of cases there is a mental background of fear and the physical condition is often poor.

*Clinical Findings* The patient is usually a young adult who complains of breathlessness, sighing, palpitations, fatigue, sweating, nervousness, dizziness, and left inframammary pain.

*On Examination*. No signs of organic disease are found. The physique is often poor, there is vasomotor spasm of the extremities as evidenced by cold or bluish red hands and feet, and abnormal sweating is seen in the axillæ and on the hands and feet. The heart. The apex beat may be forcible but there is usually no cardiac enlargement. The heart sounds are normal but the first sound may be accentuated if there is tachycardia. A short soft systolic murmur may be heard at the apex or base. The average resting pulse rate is between 80 and 100. The blood pressure is usually normal. The exercise tolerance may be impaired but is often normal.

*Differential Diagnosis* Such conditions as rheumatic carditis, early mitral stenosis, thyrotoxicosis, pulmonary tuberculosis, pleurisy, angina pectoris and malingering must be excluded. A carefully taken history will indicate the correct diagnosis in the majority of cases, and this is confirmed by the characteristic symptoms and signs.

*Prognosis* This is unfavourable, only about 25% of soldiers suffering from the syndrome can be rendered fit for full military duties. Medical boards should not enlist neurotics who will never make efficient soldiers.

*Treatment* When so enlisted, however, an attempt should be made to improve their condition. Properly graduated exercises should be first given to increase the cardio-respiratory efficiency. The patient should also be informed of the nature of his complaint and treated as a psychoneurotic.

### Simple Bradycardia

**Etiology.** Simple bradycardia may be due to vagotonia, occurring during convalescence from severe illnesses such as influenza or typhoid fever. It is also met with in association with starvation, nervous exhaustion, cerebral abscess, tumour or hæmorrhage, meningitis, myxædema, jaundice, uræmia, vaso-vagal attacks, overdosage of digitalis, and at times with myocardial degeneration. Irritation of the vagus by a mediastinal tumour may also cause bradycardia. Physiological bradycardia is met with in tall athletes, in adolescence, and old age.

**Clinical Findings.** The apex rate varies between 40 and 60. The electrocardiogram shows no evidence of heart-block.

**Treatment.** No special treatment is required beyond that indicated for the underlying condition.

### Sinus Arrhythmia

#### *(Physiological or Juvenile Cardiac Irregularity)*

**Etiology.** Sinus arrhythmia is met with in young people and during convalescence from febrile illnesses. It is due to alteration of vagal tone with respiration.

**Clinical Findings.** The patient is usually a young adult. Sinus arrhythmia generally causes no symptoms, but when discovered it may be mistaken for some serious cardiac irregularity. The pulse rate is found to slow during expiration and quicken during inspiration. This may occur periodically and only be detected when the patient is asked to breathe slowly and deeply. The irregularity disappears when the heart beats rapidly, as after exercise. The electrocardiogram shows that the alteration is due to variations in the length of diastole.

**Treatment.** Sinus arrhythmia is of no pathological import, and requires no treatment.

### Paroxysmal Tachycardia

The following varieties are described: 1. Simple paroxysmal tachycardia. 2. Nodal tachycardia. 3. Ventricular tachycardia. 4. Paroxysms of auricular flutter. 5. Paroxysms of auricular fibrillation.

#### Simple Paroxysmal Tachycardia

**Etiology.** The paroxysms of tachycardia are probably due to impulses arising at a new focus in the auricle, constituting a regular series of premature systoles. Some cases are associated with pregnancy, others remit during pregnancy. Exertion, emotion or flatulence may induce an attack.

**Clinical Findings.** The patient complains of periodical attacks of palpitations, which may be accompanied by faintness, dyspnoea and præcordial distress.

**On Examination:** The patient may be pale, somewhat cyanosed and sweating. The apex and pulse rate vary usually between 140 and 230. The rhythm is regular and is not slowed on lying down. The systolic blood pressure falls and the output from the heart is diminished.

In severe cases signs of cardiac decompensation are evident such as dilatation of the heart, oedema of the lungs and legs, and engorgement of the liver. The electrocardiogram shows a rapid regular rhythm, with normal ventricular complexes. The P wave is often inverted in leads II and III and modified in lead I.

**Differential Diagnosis** The sudden onset and cessation, and the constancy of the rate with exercise, rest and change of posture differentiate it from simple tachycardia. The engorgement of the liver may suggest an abdominal lesion, or the signs at the bases of the lungs may simulate pneumonia. The history, signs, and electrocardiogram establish the diagnosis.

**Course and Complications** The attack lasts from a few minutes to two weeks and then stops abruptly. Blindness and epileptiform convulsions may occur temporarily. Death during an attack has been recorded in a few cases.

**Treatment** The patient should lie down. The following devices may be employed to abort the attack. Holding the breath, pressure over both carotid sinuses, pressure on the eyeballs, a tight abdominal binder, ice applied to the precordium, swallowing a hard article of food such as a crust of bread or the induction of vomiting by faucial irritation. If the paroxysm is persistent quinidine should be given as for auricular fibrillation (see p. 217). An attack may sometimes be averted at the earliest premonitory symptom by taking 1 Doryl tab (carbacholum B.P. Add) nig 2 by mouth. In some cases camphor monobromata gr 3 as a pill, or a mixture containing Tne sumbul m 10 sod bicarb gr 15 sp chlorof m 7 infus gent co rec ad fl oz I is successful, when other measures fail.

### Nodal Tachycardia

Paroxysms of premature systoles arise in the A-V node, resulting often in a simultaneous contraction of the auricles and ventricles. In the electrocardiogram the P-R interval may be shortened and the P wave inverted or the P and R waves are fused, or the P wave may follow the R wave. The clinical picture and treatment resemble those of simple paroxysmal tachycardia.

### Ventricular Tachycardia

This is a rare condition due to a regular sequence of premature ventricular systoles. The ventricles may contract 180 times a minute, while the auricular rate remains at about 80. It is usually associated with severe myocardial damage, coronary occlusion or gross digitalis poisoning. The clinical features closely resemble those of the other varieties of simple paroxysmal tachycardia, but the ventricular beats are often slightly irregular. Treatment consists in giving quinidine gr 5 by mouth every 6 hours, but it is not always effective and the outlook is serious.

### Paroxysms of Auricular Flutter

This is a common cause of paroxysmal tachycardia. The apex and pulse rate are about 160, the rate is uninfluenced by posture and the

rhythm is usually regular. The auricular rate is about 200 to 300. Diagnosis is established by means of the electrocardiogram. The treatment is as for auricular flutter (see p. 219).

### Paroxysms of Auricular Fibrillation

The apex and pulse show the typical irregularity of auricular fibrillation (see p. 216) and the electrocardiogram confirms the diagnosis. The treatment consists in the administration of quinidine as for auricular fibrillation (see p. 217).

### Premature Systoles

#### (*Extra Systoles*)

**Definition.** Interpolation on the normal sinus rhythm of premature contractions arising at some other focus in the heart than the sino-auricular node.

**Etiology.** Premature systoles are believed to be due to undue excitability of the auricle, A-V node or ventricle. They are commonly met with in middle age, associated with myocardial degeneration. In young people they may occur without any evidence of cardio-vascular lesions. They may be associated with oversmoking, neurasthenia, the administration of digitalis or aconite, or coal-gas poisoning. There are three main varieties, auricular, nodal and ventricular. In about 70% of cases they are ventricular in origin.

**Clinical Findings.** The patient may be quite unaware of any cardiac irregularity, or he may be conscious of a pause in the cardiac rhythm, as if the heart had missed a beat. This is particularly noticeable when the patient is in bed. He may also complain of palpitations.

**On Examination:** The pulse may either show a pause due to the premature beat being of insufficient force to reach the wrist (intermittent pulse), or a weak beat may be felt. On listening over the apex a premature and weak contraction will be heard at the moment that the beat is missing or weak at the wrist. An occasional premature contraction may occur, or they may be frequent, causing a gross irregularity. *Pulsus bigeminus*, which may be met with in overdosage of digitalis, consists of coupled beats due to premature systoles. The electrocardiogram will show typical curves and indicate the site of the irregularity.

**Differential Diagnosis.** Premature systoles must be diagnosed from heart-block or a slow auricular fibrillation. In heart-block, when the pause is felt at the wrist, there is silence on auscultation over the apex. Premature systoles are usually diminished in frequency or abolished by exercise, but with a slow fibrillation it may be impossible to establish the diagnosis without an electrocardiogram.

**Prognosis.** Premature systoles can usually be disregarded in young people. Later in life, especially if there is evidence of cardio-vascular degeneration, they are generally indicative of myocardial degeneration.

**Treatment.** Young people should be assured that there is no cause for worry. The patient should not smoke. Quinidine gr. 3 t.i.d.s. will often ensure a regular rhythm. If there is real distress, particularly at night, ammon. brom. in doses of gr. 5 to 10 should be given t.i.d.

## Auricular Fibrillation

*(Delirium Cordis Pulsus Irregularis Perpetuus The Mitral Pulse)*

**Definition** A condition characterised by flickering auricular contractions of a peculiar type with an irregular ventricular response

**Etiology** Auricular fibrillation may be associated with infections, especially with rheumatism and less frequently with scarlet fever, diphtheria influenza and pneumonia Mitral stenosis is often associated with it Later in life it may be a degenerative phenomenon It may occur also in Graves' disease and in hypertensive heart disease

**Pathology** It is believed that in auricular fibrillation the excitation wave circulates around the orifices of the venæ cavæ, pursuing an uneven course in the auricle which is constantly altering from point to point This is known as the 'circus movement' About 450 such circulations occur each minute but the ventricle responds irregularly to the varying number of stimuli it receives through the A-V node

**Clinical Findings** The disturbance produced varies with the frequency of ventricular contractions If there is a slow fibrillation there may be no subjective symptoms but when the ventricle contracts rapidly, cardiac decompensation usually ensues The patient may complain of palpitations, or of præcordial discomfort, dyspnoea and swelling of the extremities

**On Examination** The patient may or may not be dyspnoeic at rest or on slight exertion, according to the degree of heart failure present There is usually some cyanosis of the lips or ears The heart Inspection The impulse is usually visible, the apex being displaced a little downwards and outwards Palpation The apex beat is forcible and a diastolic thrill may be felt, although this may disappear with the onset of fibrillation Percussion The area of cardiac dulness is often increased both to the right and to the left Auscultation The first sound at the apex is usually forcible, and an early or mid diastolic murmur may be heard if mitral stenosis is present The presystolic murmur of mitral stenosis disappears with the onset of fibrillation if the heart is beating slowly The rhythm is characteristically completely irregular, the beats also varying in intensity, so that many of the ventricular contractions fail to cause a pulse at the wrist. The pulse This is completely irregular and its rate is usually slower than that of the ventricle Records of pulse rates are therefore quite valueless in determining the rate of the heart The lungs liver, abdomen and extremities may show signs of venous stasis and œdema (see p. 226) The electrocardiogram is typical the P waves are absent, and the ventricular complexes are irregular in rate and degree

**Differential Diagnosis** Auricular fibrillation can usually be readily diagnosed by examination of the heart and pulse Without a tracing, slow fibrillation is difficult to differentiate from premature systoles.

**Course and Complications** Auricular fibrillation may occur in paroxysms the normal rhythm being restored spontaneously from time to time In other cases, and despite treatment, it persists for indefinite periods, or the normal rhythm may be quickly restored by adequate



treatment. Complications include congestive heart failure, and systemic embolus formation. The latter is especially liable to follow restoration to normal rhythm by the administration of quinidine, a portion of clot being detached from the left auricle. The main arterial supply to a limb may be obstructed, with severe pain. The limb becomes cold and blue, and gangrene may supervene.

**Prognosis.** This is always grave, although a patient may live for over 10 years with auricular fibrillation.

**Treatment.** The effective mode of digitalis administration is often not appreciated, and disappointing results are then obtained. In cases in which the apex rate is over 100, or if there are signs of heart failure, the patient should be put to bed and digitalisation effected. It may be taken that fl. oz.  $\frac{1}{2}$  of a good tincture of digitalis will be required to produce the full effect in a male weighing 10 stones, and m. 180 for a man weighing 8 stones. The drug should not be given more often than every 6 hours. Thus m. 10 of the digitalis can be given six-hourly for 4 doses, and then m. 30 six-hourly until the required amount has been administered. The apex rate will then generally have slowed to about 90. No more should now be given for a day or so, when a dose of m. 20 t.i.d. can be given. The minimum amount required to keep the apex rate at about 70 to 80 should then be determined. Symptoms of over-dosage of digitalis are undue slowing of the apex rate (below 60), coupling of beats due to a regular sequence of premature systoles, the onset of pulsus alternans, diminution in the output of urine, and vomiting. Vomiting at the onset of the treatment is usually due to congestion of the gastric mucosa, and not to the digitalis. The urinary output should always be measured while the patient is taking large doses of digitalis. If the patient vomits the tincture, other preparations can be employed. Tablets of digitalis pulv. (B.P. Add.) gr. 1 (equals m. 10 of the tincture) are sometimes preferred. Digoxin, a crystalline substance, can be given orally or intravenously. The oral tablets contain 0.25 mg. The initial oral dose is 1.5 mg. for a patient weighing 10 stones or over, and 1 to 1.25 mg. for lighter patients. Six hours later 0.25 mg. is given, and this is repeated every 6 hours until the ventricular rate falls to 70 or 80. Subsequently a maintenance dose of 0.25 mg. once or twice a day may be required. In very urgent cases an intravenous injection of 1 mg. of digoxin can be given. 0.5 mg. is put up in 1 mil. of 80% alcohol. This is diluted with nine times its volume of sterile saline and injected slowly. Two hours later 0.25 mg. of digoxin can be given by mouth. In desperate cases strophanthin gr. 1/200 or ouabaine (a preparation of g-strophanthin) gr. 1/240 should be injected intravenously.

**The Use of Quinidine.** This may be given in selected cases, with the object of restoring normal rhythm. The main contra-indications to the use of quinidine are myocardial degeneration, a history of recent embolus, heart failure or heart-block. The most suitable cases are those which are inflammatory in type and recent in origin, or those occurring in association with hyperthyroidism. It is usually considered inadvisable to administer digitalis and quinidine simultaneously. The patient must be kept in bed. A test dose of gr. 3 (0.2 G.) of quinidine

in a gelatin capsule is given. If no toxæmic symptoms appear, such as sweating, nausea, vomiting, diarrhœa, abdominal pain, a scarlatiniform rash, dimness of vision or severe headache, the administration can be proceeded with. On the next day, gr 6 (0.4 G) quinidine are given every 3 hours for 4 doses, and the day after gr 6 quinidine are given three hourly for 5 doses. This may be continued for another 4 days. The pulse, and if feasible, the apex should be examined before each dose is given, and if found regular the quinidine should be discontinued. The occurrence of frequent premature systoles is also an indication for omitting the drug. If a regular rhythm is not restored at the end of a week on the above dosage the quinidine will probably have no effect, and should be stopped. In cases in which the normal rhythm has been restored it may be necessary to give a daily dose of gr 5 for long periods. When arterial embolism occurs the artery should be immediately incised and the clot removed.

### Auricular Flutter

**Definition** A condition characterised by rapid and regular auricular contractions of a peculiar nature, and almost invariably accompanied by heart block.

**Etiology** Auricular flutter is usually associated with myocardial degeneration and arteriosclerosis, much less frequently with infective diseases or with thyrotoxicosis.

**Pathology** It is believed that a wave circulates around the orifices of the venæ cavæ, and tangential waves spread from the mother wave to distant parts of the auricles. This is a form of "circus movement." The auricular rate is about 200 to 350 contractions a minute, and the ventricle contracts about 150 times a minute.

**Clinical Findings** The patient is usually a male past middle age. He gives a history of attacks of palpitations, usually sudden in onset and in cessation, but the last attack may have persisted.

**On Examination** The heart is usually enlarged and the arteries are thickened. A valvular lesion may or may not be present. The pulse and apex rate are generally between 130 and 160, the rhythm is regular, and the rate is unaffected by posture and by exercise. In the electrocardiogram regularly recurring dome shaped P waves are seen with ventricular complexes occurring at regular or irregular intervals, but less frequently than the P waves, owing to heart block.

**Differential Diagnosis** Paroxysms of flutter can be differentiated from simple paroxysmal tachycardia by suitable tracings. Auricular fibrillation may be simulated if in flutter the ventricular responses are irregular. Slight exercise, however, in flutter, usually renders the ventricular rhythm regular, with a definite grade of heart block, such as 3:1 or 2:1. This does not occur in auricular fibrillation.

**Course and Complications** Paroxysms of flutter may occur, or a continuous stage may persist for as long as 10 years. Paroxysms may ensue, in which the ventricle assumes the auricular rate. These are very dangerous, the patient rapidly losing consciousness and dying,

if the heart-block is not restored quickly. Congestive heart failure may occur as a complication of flutter.

**Prognosis.** This is always serious, but not usually immediately grave. The condition of the myocardium is a factor of great importance, although hard to estimate.

**Treatment.** The patient should be put to bed and digitalised, as for fibrillation (see p. 217). This usually converts the flutter rhythm to that of fibrillation. On stopping the digitalis a normal rhythm may ensue. If the patient will not tolerate digitalis, an intravenous injection of strophanthin gr. 1/200 may be given, or a course of quinidine, as described on p. 217. The normal rhythm may then be restored direct, without an intermediate stage of fibrillation.

### Ventricular Fibrillation

This is probably the cause of sudden death in coronary obstruction and chloroform anaesthesia. It is compatible with life only if the fibrillation is of very short duration.

### Heart-block

**Definition.** A condition characterised by delay in the conduction of impulses along some portion of the junctional tissue of the heart. The following varieties are described: 1. Sino-auricular block. 2. Auriculo-ventricular block. 3. Bundle-branch block. 4. Arborisation block.

**Etiology.** Congenital heart-block is due to a defect, such as a patent interventricular septum, interrupting the bundle of His, or rarely to aorto-pulmonary patency. Acquired heart-block may be due to inflammatory lesions caused by rheumatism, diphtheria, influenza, pneumonia, and enteric or scarlet fever, or to degenerations, especially that caused by syphilis. It may also result from overdosage with digitalis, strophanthin, squills, or quinidine. A gumma, tumour, cyst, or an area of fibrosis or atrophy may be the causative factor in some cases.

### Sino-auricular Block

#### (Tortoise Heart)

The impulses arising in the S-A node at times fail to provoke an auricular contraction. The whole heart then misses a beat, but the succeeding contraction occurs at approximately the normal interval. The ventricle may occasionally interpolate a beat on its own ("ventricular escape"), when the heart is beating at the slow rate. It is a cause of dropped beats, and can only be distinguished from auriculo-ventricular block by a tracing, the complete absence of the P.R.T. deflections in the electrocardiogram corresponding with the pauses in the heart beats. It is probably of no clinical importance, but if occurring regularly every other beat the pulse is slow, about 50. Exercise usually causes the rate to double and the restoration of normal rhythm can also be effected, except in cases of long standing, by the administration of atropin. sulph., gr. 1/200 in 1 fl. oz. of water t.i.d.

## Auriculo-ventricular Block

Delay occurs in the passage of the impulse from the auricle to the ventricle. Four grades may be recognised :—

*Grade I* This is the earliest stage, and can only be detected by a tracing. The electrocardiogram shows a prolongation of the P-R interval to more than the normal of  $\frac{1}{2}$  second. *Grade II*. The ventricle occasionally fails to respond to the auricular impulse. *Grade III*. The ventricle fails in a regular manner to respond to the auricular stimuli. Thus every fourth ventricular beat may be missing (4 : 1 block) or other sequences such as 3 : 1 or 2 : 1 block may be present. *Grade IV*. There is complete dissociation between auricle and ventricle, the ventricle contracting regularly at its own independent rate of about 30 to 40 a minute. The electrocardiogram allows all these grades of block to be diagnosed with certainty.

**Clinical Findings.** The patient does not usually notice anything abnormal except in the severe degrees of heart-block. Attacks of unconsciousness (Adams-Stokes syndrome) are liable to occur if the A-V bundle fails to conduct the impulse and the ventricle does not take up its independent rhythm. The attacks come on suddenly with convulsive movements of the face and arms, usually the tongue is not bitten, and there is no involuntary micturition. The patient is pale and falls down, he then becomes cyanosed and the breathing is stertorous. Unless the ventricle begins to beat again, he will die.

**On Examination** Auscultation at the apex of the heart will show that the heart misses a beat, when a beat is dropped at the radial pulse. A ventricular rate below 10 a minute is almost always due to complete heart block. It is often possible to see pulsation in the jugular veins corresponding in rate with the auricular contractions, and so faster than the apex beat or radial pulse.

**Differential Diagnosis.** Clinically the missed beats which occur at the wrist in heart-block must be distinguished from those resulting from feeble premature systoles. In the former, as described above, there is silence on listening over the heart, when the beat is absent at the wrist. Inhalation of amyl nitrite in 2 : 1 heart-block will often suddenly double the rate of the pulse, the apex subsequently abruptly reverting to its slow rate. This change does not occur in simple bradycardia. The ventricular rate in complete heart-block is not affected by exercise or by the inhalation of amyl nitrite, or by the injection of atropine sulphate.

**Course and Complications.** Heart block, especially in the young, when arising during the course of, or in convalescence from an acute illness, is usually a temporary derangement. If due to a degenerative lesion, it is likely to be permanent. With the stage of onset of complete block, the Adams-Stokes syndrome is to be feared.

**Prognosis.** Heart-block always indicates some degree of myocardial abnormality, due either to an inflammatory lesion, which may be temporary, or to a permanent degenerative one. The outlook is more serious when complicated by Adams-Stokes attacks.

**Treatment.** When heart-block develops during the course of an

infective illness, the patient should be kept strictly at rest in bed until the normal rhythm is restored, or until it is considered that the heart-block is permanent. In other cases the Wassermann reaction should be determined, and, if positive, a course of potassium iodide and mercury given (see p. 248). For Adams-Stokes attacks subcutaneous injection of m. 8 of liq. adrenal. hydrochlor. should be given immediately, followed by ephedrine sulphate gr.  $\frac{1}{2}$  t.d.s. by mouth. If the heart has stopped beating, an intracardiac injection of m. 8 of liq. adrenal. hydrochlor. should be given. An alternative method of treatment is to give barium chloride gr. 1 t.d.s. by mouth.

### Bundle-branch Block

There is delay in conduction in one or other of the branches of the bundle of His, leading to the left or right ventricle. This condition can only be diagnosed by means of an electrocardiogram. It is of grave significance, death often occurring within two years of its detection, although some patients live for several years after its diagnosis.

### Arborisation Block

The terminal subendothelial divisions of Purkinje's fibres have impaired conductivity. This lesion can be detected by an electrocardiogram. The prognosis is usually bad.

### Pulsus Alternans

**Definition.** Alternate strong and weak contractions of the ventricles.

**Etiology.** Pulsus alternans is probably a manifestation of myocardial degeneration, the heart labouring against an excessive burden; it may also result from an overdose of digitalis.

**Clinical Findings.** Pulsus alternans is difficult to detect by the finger. A radial tracing will, however, show alternate large and small beats at very nearly regular intervals. If the blood pressure is taken by the auscultatory method, only alternate beats will be heard at higher pressures, whereas on lowering the pressure in the armlet each beat will be audible. An electrocardiogram may show R waves of equal intensity, although the radial tracing shows definite alternation. Pulsus alternans can be differentiated from premature systoles regularly recurring with every other beat (pulsus bigeminus) by the short interval between the normal and successive premature beat, and the longer interval between the premature beat and the next normal one. The condition is of grave import when occurring in a pulse of normal rate, but if associated with paroxysmal tachycardia or with digitalis medication the prognosis is not so serious.

### Pulsus Paradoxus

**Definition.** A condition in which the pulse weakens in intensity with inspiration and becomes stronger with expiration.

**Etiology.** Pulsus paradoxus is often associated with mediastino-pericarditis, and with pericardial effusion, and may be explained by the fact that normally during inspiration the roots of the lungs descend and

carry the heart with them. If, however, the heart is tethered by adhesions, or pressed on by a pericardial effusion, during inspiration it is pulled on from above and below. The orifices of the *venæ caviæ* will then be partially obstructed, with diminished cardiac inflow and output.

### THE MYOCARDIUM

The pathological affections of the myocardium may be grouped as follows: *Atrophy*, *Cloudy swelling*, *Fatty degeneration* and *infiltration*, *Fibrosis*, *Amyloid*, *hyaline* and *calcareous degenerations*, *Granulomata*, *Tumours*, simple and malignant, *Cysts*, *Hypertrophy*, *Dilatation*, *Inflammation*, *Vascular degeneration* and *infarct*, *Rupture*.

*Atrophy*. This is usually a brown atrophy. It is common in old age and in wasting diseases, such as tuberculosis and cancer. *Cloudy swelling*. This is met with in acute fevers and septicæmia. *Fatty degeneration*. This occurs in acute fevers, diphtheria, alcohol, chloroform or phosphorus poisoning, and in blood diseases, such as *anæmia* and *leukæmia*. It causes the change known as 'tabby cat' striation or thrush's breast appearance, seen under the endocardium, usually in the left ventricle. *Fatty infiltration*. This is sometimes but not invariably, associated with obesity. *Fibrosis*. This is associated with arteriosclerosis and narrowing of the coronary arteries. The areas of fibrosis may be localised or diffuse and are commonly found in the left ventricle. It may occur diffusely, as a sequel of rheumatic carditis. *Amyloid*, *hyaline* and *calcareous degenerations*. These are less common. *Granulomata*. These include syphilis and tuberculosis of the heart. *Tumours*. A lipoma or fibroma may develop or secondary melanotic sarcoma or rarely secondary carcinoma. *Cysts*. A hydatid may form in the heart. *Hypertrophy*. This is associated with valvular disease such as aortic or mitral regurgitation, with adherent pericardium, increased blood pressure (hypertensive heart disease), thyrotoxicosis, chronic nephritis and arteriosclerosis. The normal weight of the heart is 0.4% of the body weight. Congenital idiopathic hypertrophy is a rare condition, which usually results in death in less than a year. *Dilatation*. This may be due to fatty degeneration to acute inflammatory changes, or it may be associated with hypertrophy. *Inflammation*. Acute simple myocarditis. This occurs in infections, especially rheumatic fever (see p. 577), and diphtheria and less often with enteric or scarlet fever, septicæmia, Graves disease and syphilis. Acute suppurative myocarditis. Small embolic abscesses may form in the myocardium in pyæmia as in puerperal fever, osteomyelitis or ulcerative endocarditis. *Vascular degeneration and infarct*. These are especially associated with coronary obstruction (see p. 230). *Rupture*. This may follow an infarct or result from penetrating injuries or occasionally from non penetrating injuries of the chest wall.

The following clinical varieties of myocardial affections will be described: The hypertensive heart, the cor pulmonale, the failing heart, coronary obstruction and non penetrating injuries of the heart.

The fatty heart and the fibroid heart do not justify separate clinical descriptions. Sudden death from fatty heart is due probably to ventricular fibrillation, and not to the fat around the heart. Fatty degeneration of the heart, however, such as occurs in diphtheria, leads to cardiac dilatation, tic-tac rhythm, and irregularities such as heart-block or gallop rhythm. It is a very fatal condition. Fibroid heart is often described as a condition of hypertrophy of the left ventricle, such as is found in hypertensive heart disease.

### Hypertensive Heart Disease

**Definition.** Hypertrophy of the left ventricle due to hyperpiesia.

**Etiology.** The cardiac enlargement is secondary to the high blood pressure. The cause of the high blood pressure (essential hypertension) is unknown, but the subject is discussed on p. 266.

**Pathology.** There is hypertrophy of the heart. The left ventricle is chiefly affected. The muscle fibres are increased in size.

**Clinical Findings.** The patient is usually an adult over the age of 40. He complains of symptoms due to high blood pressure (see p. 267). No cardiac symptoms are noticed until the reserve of cardiac power is overtaxed, or some complication such as disturbance of coronary circulation occurs. Symptoms include shortness of breath on exertion, attacks of cardiac asthma, palpitations or præcordial pain.

**On Examination:** The heart. The apex beat is usually displaced downwards and outwards. A systolic murmur may be heard at the apex, due to mitral regurgitation and occasionally there is an aortic systolic or diastolic murmur. With failure of the heart, pulsus alternans (see p. 221) or gallop rhythm (see p. 225) may be present. The blood pressure, which is raised in the early stages of the disease, often falls with the onset of failure of compensation, although the diastolic figure generally remains over 100 mm. Hg. Ophthalmoscopic examination may show retinal arteriosclerosis.

**Differential Diagnosis.** Hypertensive heart disease must be differentiated from cardiac enlargement associated with chronic nephritis, or due to aortic disease or thyrotoxicosis. The diagnosis is difficult in the later stages, especially if the blood pressure has fallen.

**Course and Complications.** The course is progressive. Complications include congestive heart failure, cerebral hæmorrhage, angina pectoris, coronary thrombosis, and rarely uræmia. Intercurrent infections are not uncommon.

**Prognosis.** This is always unfavourable, especially when there is marked cardiac enlargement, retinal arteriosclerosis, or myocardial failure.

**Treatment.** This is as described for hyperpiesia and for congestive failure (see pp. 267, 227).

### Cor Pulmonale

#### (Pulmonary Hypertension)

**Definition.** Enlargement of the right ventricle, due to obstruction in the pulmonary circulation or at the mitral valve.

**Etiology** Pulmonary hypertension is due to such causes as emphysema, pulmonary arteriosclerosis (Ayerza's disease, see p 264), pulmonary fibrosis or mitral stenosis. Acute cor pulmonale may occur with a pulmonary embolus which causes a severe degree of occlusion of the pulmonary artery. There is sudden dilatation of the right auricle and ventricle.

**Pathology** There is first hypertrophy of the right ventricle, with secondary dilatation of the right ventricle, the tricuspid valve, right auricle and pulmonary artery. There may also be atheroma of the pulmonary artery.

**Clinical Findings** The patient only complains of symptoms when there is failure of the right ventricle. There is then increased dyspnoea and cough. In acute cor pulmonale the symptoms are those of pulmonary embolus.

**On Examination** In some cases it is possible to detect by percussion that the right auricle is enlarged, often it is obscured by an emphysematous lung. The pulmonary second sound is usually accentuated. The blood pressure is low, such as 110/80 mm Hg. With right sided failure the veins in the neck are engorged, the liver enlarges and often there is ascites and oedema of the legs. The right sided hypertrophy may be detected by X ray examination or by an electrocardiogram, and a dilated pulmonary artery may be revealed radiographically. In acute cor pulmonale there is cyanosis, with engorgement of the veins in the neck. The pulmonary second sound is accentuated and gallop rhythm may be present.

**Treatment** The patient should be rested in bed and treated with digitalis, as for congestive heart failure (see p 227).

## The Failing Heart

### (Congestive Heart Failure)

**Pathogenesis** The exact nature of heart failure is still uncertain. The back pressure theory was generally accepted in the middle of the last century. According to this left ventricular failure causes dilatation and incompetence of the mitral valve, with resultant congestion of the lungs and subsequently, secondary failure of the right ventricle and dilatation of the tricuspid valve lead to systemic congestion. Later, the forward theory was introduced, heart failure being considered as due essentially to inefficiency of the driving force of the heart muscle, with in consequence deficient blood supply to the tissues. This theory was largely based on observations made on heart failure in auricular fibrillation which often affects both sides of the heart simultaneously. We have now returned to the older view that the symptoms of heart failure are mainly due to congestion occurring in the circulatory territory immediately behind the side of the heart which first fails but in some cases deficient cardiac output also plays its part. Heart failure may thus affect either side of the heart independently, or both sides may be involved simultaneously. Further, such failure may be acute or insidious in onset. From what has been said above, it will be clear



that we should expect to find systemic congestion with right-sided failure and pulmonary congestion with left-sided failure.

**Etiology.** 1. *Left heart failure.* This is approximately three times as common as right heart failure. The important causes are hypertensive heart disease, aortic stenosis, aortic regurgitation, chronic nephritis, and coronary occlusion. 2. *Right heart failure.* This is most frequently secondary to left heart failure. Other important causes are mitral stenosis, pulmonary embolus or thrombosis, pulmonary arteriosclerosis and pulmonary stenosis. Pulmonary diseases such as emphysema, with or without associated kyphoscoliosis, pulmonary fibrosis and pneumoconiosis are not now considered frequent or important causes of right heart failure. 3. *Left and right heart failure.* This may be associated with rheumatic carditis, diphtheria, severe anæmia, generalised coronary arteriosclerosis, auricular fibrillation, auricular flutter, paroxysmal tachycardia, hyperthyroidism and constrictive pericarditis.

Heart failure may be acute or insidious; for example, acute left heart failure may follow hypertension, aortic disease or coronary occlusion, and acute right failure may be associated with pulmonary embolus, thrombosis or lobar pneumonia.

**Clinical Findings.** *Left heart failure.* Three stages are described: (a) Paroxysmal pulmonary congestion. Attacks of acute œdema of the lungs or of nocturnal dyspnoea occur, with cough and lesser degrees of pulmonary œdema. (b) Pulmonary congestion of effort. Here shortness of breath, cough and often blood-stained sputum are provoked by exercise, excitement or a cold atmosphere. (c) Chronic pulmonary congestion. This is characterised by shortness of breath and at times hæmoptysis. Such cases are liable to be mistaken for chronic bronchitis, pulmonary tuberculosis or a new growth.

The chief clinical features of left heart failure are pallor with some cyanosis, and dyspnoea especially on effort and at night. The heart is usually enlarged to the left, and gallop rhythm or pulsus alternans may be present (see p. 221). With gallop rhythm a third heart sound is heard during mid or late diastole, the heart sounds are then likened to the sound produced by saying "lub lub dupp." Graphic records indicate that the first part of the first sound occurs in presystole and is presumably auricular in origin. It is also known as presystolic gallop, the extra sound perhaps being due to vibrations of a rapidly filling ventricle which lacks tone. It does not occur in auricular fibrillation. This third sound can also be felt as a diastolic impulse. The three heart sounds are evenly spaced, and the third sound is only heard when the rate is rapid. It is of very grave significance. Presystolic gallop must be distinguished from protodiastolic gallop. In the latter a third sound closely follows the second sound, "lub dupp dupp." This is of no significance, being an accentuation of the physiological third heart sound. It is best heard near the apex of the heart, whereas a split second sound is most noticeable near the base. The blood pressure is usually high, especially the diastolic figure, and the rhythm is usually regular, but premature systoles or paroxysmal auricular fibrillation may occur. Râles are heard at the bases of the lungs and a hydrothorax

may develop, more frequently on the left side, and, if bilateral, it is usually larger on the left side. It is sometimes interlobar. The electrocardiogram often shows left axis deviation with inverted  $T_1$ , or  $T_1$  and  $T_2$  waves, or there may be bundle branch block. X ray examination shows that the pulmonary arterial shadows are unduly dense and enlarged, and a blurred zone around the pulmonary roots indicates the onset of pulmonary oedema. If right-sided failure now supervenes the symptoms of pulmonary congestion are, to a certain extent, relieved, and paroxysms of dyspnoea usually cease. The attacks of nocturnal dyspnoea are often known as *cardiac asthma* and they may be associated with bronchial spasm. The patient wakes up suddenly, feels suffocated, sits up, struggles for breath, and finally he may fall back exhausted and sweating. The attacks may be more severe, the patient having acute oedema of the lungs (see p 173). Cheyne-Stokes breathing is seen in other cases, the breathing waxing and waning, with intervals of apnoea lasting 30 or 40 seconds. During the hyperpnoeic period which results from stimulation of the respiratory centre,  $CO_2$  is washed out of the blood. This produces the apnoeic phase. The patient may sleep in the apnoeic phase and wake with each period of hyperpnoea. Cardiac dyspnoea is not now believed to be due to deficient supply of blood to the respiratory centre or to anoxia of the centre, but rather to a nervous reflex which originates in the lungs and passes to the respiratory centre through the vagus. Diminished elasticity of the lungs due to congestion is thought to originate the reflex. When the patient is lying down at night the vital capacity is diminished and pulmonary congestion increases, probably owing to increased output from the right ventricle and possibly to a further and sudden dilatation of the left ventricle.

*Right heart failure* (see also Cor Pulmonale, p 223). The clinical findings vary with the severity of the condition and are dependent upon systemic congestion. The face is cyanosed, there is dyspnoea on exertion, palpitations, lassitude and distress or pain in the præcordium. As the condition progresses the dyspnoea becomes more noticeable. The pulse is frequent, the blood pressure tends to be low. The heart dulness is enlarged to the right and left if it is not obscured by emphysema. This is due to the right auricular dulness extending to the right, and the right ventricular dulness to the left. A systolic murmur is sometimes audible over the tricuspid area. An electrocardiogram will show right axis deviation and a radiogram reveal enlargement of the right auricle and right ventricle. Paroxysms of cardiac dyspnoea do not occur with right-sided failure although spasmodic asthma may be noted. In advanced cases the veins in the neck are engorged, the liver is enlarged, tender and pulsating, and there is oedema of the extremities, and ascites. In addition a transudate forms in the pleural sacs, usually more so on the right side, and there may also be hydropericardium. There is albuminuria with diminished output of urine. Other features which may be noted include lack of concentration, giddiness, fainting attacks, nausea and vomiting, diarrhoea, abdominal pain, jaundice and hæmaturia.

The determination of the arm to-tongue circulation rate and of the

venous systemic blood pressure affords further evidence of left or right ventricular failure. With left ventricular failure the arm-to-tongue circulation time is increased but the systemic venous pressure is usually normal; whereas with right ventricular failure both the circulation time and the venous pressure are increased.

*The circulation rate.* The arm-to-tongue circulation rate is used to determine the rate of circulation through the lungs. Five mils of a 20% solution of Decholin (sodium dehydrocholate) are injected through a wide-bore needle into an antecubital vein of the right arm, the injection taking 2 to 3 seconds. The patient sits propped up at an angle of 45° and a local anæsthetic is used to prevent tachycardia resulting from pain of the injection. The patient is told to raise his left hand immediately he notices a bitter taste under the tongue. The time is taken with a stop watch from the beginning of the injection of the Decholin to the signal that it has reached the tongue. The normal time is between 10 and 17 seconds, and, with both right and left heart failure, it may be increased up to 40 seconds or more.

*The systemic venous blood pressure.* This can be gauged clinically by observing the cervical veins with the patient recumbent and propped up. Normally the venous pressure is zero at the point in the vein which is in the same horizontal plane as the lower end of the sternum. With an increase of venous pressure the cervical veins on both sides will be engorged at a point which is on a plane higher than that of the manubrium zero level. An increased systemic venous pressure is almost certainly present if the cervical veins are dilated when the patient is propped up at 45°. The systemic venous blood pressure can be measured directly by inserting a short, bevelled 1 mm. bore needle into an antecubital vein of the right arm, with the patient propped at an angle of 45°. The needle is connected by a short piece of rubber tubing to the horizontal arm of an L-shaped graduated glass tube of 3 mm. bore. The whole apparatus is sterilised, and a sterile 3.8% sodium citrate solution run through it immediately before use. The blood enters the glass tube and a reading is taken of the height of the column of blood above the level of the 4th intercostal space. The normal reading is about 6 cm. of blood. Compression of the upper arm should cause the level to rise, and on releasing the pressure it should fall to approximately its previous level, indicating that there is no block in the apparatus.

*Treatment. Acute Left Heart Failure.* If there is œdema of the lungs an injection of morphin. sulph. gr. 1/3 and atropin. sulph. gr. 1/50 should be given. This should be followed by venesection of 15 to 20 oz. Digitalis in large doses is indicated in other cases, or to produce a rapid effect an intravenous injection of strophanthin, gr. 1/200 or of 1 mil. of Cardiazol (leptazolium B.P. Add.) should be given.

*Acute Right Heart Failure.* The patient should be put to bed and propped up. He is then bled, a pint of blood being removed from a vein in the arm. Leeches may be applied over the engorged liver. Digitalis should then be given in large doses, as for auricular fibrillation (see p. 217). Oxygen should be administered through a nasal catheter or B.L.B. mask (see p. 143) to relieve cyanosis.

*Routine Treatment of Heart Failure* The patient should be in bed, on absolute rest, propped up in the most comfortable position. He may obtain relief by leaning forward on a well padded heart table, placed across the bed. Diet The meals should be taken dry and not more than 30 oz. of fluid drunk in the 24 hours. The total calorie value should be low, 800 to 1,000 calories. A specimen diet of 810 calories is as follows—7 a.m. Tea, 1 cup, with milk 1 oz., and two level teaspoonfuls of sugar. Breakfast One egg (lightly boiled) Bread or toast, 2 slices Butter,  $\frac{1}{2}$  oz. Jam or honey,  $\frac{1}{2}$  oz. 11 a.m. Water, 8 oz. Lunch Fish, 3 oz. Green vegetables, 2 oz. Bread, 1 slice Butter,  $\frac{1}{2}$  oz. 4 p.m. Tea, 1 cup, with milk, 1 oz., and two level teaspoonfuls of sugar. 6 p.m. Water, 8 oz. Dinner Toast, 1 slice Jam or honey,  $\frac{1}{2}$  oz. Junket, 6 oz. No salt should be taken. The amount of urine passed every 24 hours should be measured and charted. Digitalis should be given in full doses, such as m 30 to 40 of the tincture every 6 hours for 1 or 2 days (see p 217), keeping watch for the onset of toxic symptoms, such as nausea, vomiting, oliguria, bradycardia or coupled beats. Digitalis should not be given if the patient is suffering from heart block. If diuresis ensues with the administration of digitalis the œdema will probably disappear. If no diuresis is provoked other measures may be tried, such as Guy's diuretic pill (pil digital co BPC), 1 t d s for 3 days, or theophyll et sod acetat gr 3, in a cup of tea t d s for 3 days, or ammonium chloride and Salyrgan. Salyrgan (mersalylum BP) which contains mercury, is put up in 10% strength in 1 ml and 2 mls ampoules. It is administered as follows. A test dose of 0.5 ml. is injected intramuscularly. If there is no intolerance, the signs of which are hæmaturia, diarrhœa or cutaneous irritation, ammon chlor gr 30 is given by mouth every 6 hours in gr 10 capsules or as four 0.5 G "stearetttes". Next day 1 ml of Salyrgan, diluted to 5 mls with normal saline is injected very slowly into a vein. The arm is then raised to prevent venous thrombosis. On the fifth day and again on the ninth day, 2 mls of Salyrgan, diluted to 10 mls with normal saline, are injected intravenously, the ammon chlor being taken by mouth every day during the course of treatment. If diuresis is not provoked after the second intravenous injection it is not usually likely that the treatment will succeed. Novurit (a mercurial theophylline compound) in doses of 2 mls, injected intravenously or intramuscularly into the upper and outer quadrant of the gluteal region, can be used as an alternative to Salyrgan, the ammonium chloride being given as above. Cardophylin (theophyll c æthylenediamina BP) can also be used as a diuretic and to dilate the coronary vessels. It can be given by mouth as a tablet (0.1 G), two to four daily, or as an intravenous injection of 0.24 G in 10 mls daily, or as an intramuscular injection of 0.48 G in 2 mls daily. A hydrothorax should be aspirated, as it severely impedes the heart's action, similarly ascitic fluid should be removed by drainage if it does not show signs of absorption. If the œdema persists in the legs it is advisable to sit the patient up in a heart chair for 12 hours and then drain the legs by making multiple small incisions,  $\frac{1}{4}$  inch long, through the skin, which has been sterilised.

with ether, on the dorsum of the feet and lower part of the front of the legs. The legs are then covered with sterile gauze, which is changed as it becomes soaked with the fluid, the legs being further protected by a cradle covered with a blanket. The gauze usually requires changing every 3 or 4 hours. Venesection should be performed for right-sided engorgement as mentioned above and continuous intra-nasal oxygen administered. Sleep should be secured either by the use of chloralamide (chloralformamidum B.P.C.) in a mixture, such as Chloralamid. gr. 30, pot. brom. gr. 15, sp. chlorof. m. 20, aquam ad fl. oz.  $1\frac{1}{2}$ , fl. oz.  $1\frac{1}{2}$  nocte; by paraldehyde m. 120 in gin fl. oz. 1 by mouth; by the rectal injection of paraldehyde fl. oz.  $\frac{3}{4}$  to 1 in fl. oz. 4 of warm saline; by Nepenthe m. 15 and aspirin gr. 10, or by a subcutaneous injection of morphin. sulph. gr.  $\frac{1}{6}$  to  $\frac{1}{4}$  with atropin. sulph. gr.  $\frac{1}{120}$  nocte. For cardiac asthma an injection of morphine should also be given. Whisky, fl. oz.  $\frac{1}{2}$  to 1, will often relieve restlessness. The bowels should be kept open freely, with the help of salines such as mag. sulph. gr. 60 to 120 mane, or pulv. jalapæ. co. gr. 40 to 60 nocte. A total thyroidectomy has produced good results in some cases of congestive failure, not associated with hyperthyroidism, which have failed to respond to other measures. Salyrgan and ammonium chloride treatment is of great value in cases of pulmonary congestion of effort and chronic pulmonary congestion, where the pulmonary congestion may be latent.

*The After Treatment.* The patient must be kept in bed until the pulse rate remains steady at about 80. He may then be allowed to sit out of bed for half to one hour daily, and subsequently gradually walk about on the level. The fluid intake should be restricted to 30 to 40 oz. in the 24 hours, and a weekly injection of Salyrgan (mersalylum B.P.) or Novurit, with the administration of ammon. chlor. gr. 30 every six hours for four days a week, may be of great benefit. During this period of convalescence massage to the extremities and the body is of great value. The walking exercise should be carefully regulated, and the patient should sit down immediately he experiences any cardiac distress.

### Peripheral Circulatory Failure

The circulation may fail primarily in the periphery rather than in the heart. This is due to slowing of the circulation caused by dilatation of the arterioles, so that there is failure of the venous return to the heart. It is met with typically in surgical shock, but may occur in acute infections such as diphtheria or pneumonia. The blood pressure is very low. The pulse is regular but very feeble, the skin is pale and cold, but the heart beat is forcible. In pneumonia the blood may be pooled in the skin, giving rise to cyanosis. Treatment of peripheral failure is often unsatisfactory. Ephedrine hydrochlor. gr.  $\frac{1}{4}$  should be given by mouth and Pitressin 1 mil. (20 units) injected intramuscularly every 6 hours, and a subcutaneous injection of Coramine (nikethamidum B.P.Add.) 1.5 mil. given t.i.d. The foot of the bed should be raised and hot bottles applied to the patient's feet.

### Syncopal Attacks

**Definition.** Unconsciousness due to deficient cerebral circulation

**Etiology.** The circulatory failure may be primarily vascular due to deficient supply of blood to the heart, or primarily cardiac due to deficient output from the heart

The chief causes are thus anæmia, low blood pressure, change of posture, vaso vagal attacks (see p 332), an over active carotid sinus reflex, heart-block and auricular flutter

**Clinical Findings** The clinical findings are described under the respective headings of the causative conditions Postural syncope does not occur when the patient is lying In all syncopal attacks, as opposed to fainting attacks of cerebral origin and not due to disordered cerebral circulation, the pulse is weak. An unduly sensitive carotid sinus reflex results in reflex vagal stimulation and slowing of the heart. It occurs especially with a raised blood pressure and arteriosclerosis Pressure over one or other carotid sinus may slow the heart and produce an attack of syncope in those predisposed

**Treatment.** This is described under the respective headings A hypodermic injection of atropin sulph gr 1/100 or of m 5 of liq adrenal hydrochlor will usually relieve vaso vagal attacks For carotid sinus syncope ephedrine sulph. gr  $\frac{1}{4}$  t.d.s by mouth may afford relief, and in severe cases denervation of the sinus has been performed.

### Cardiac Infarction

(*Coronary Thrombosis Coronary Occlusion Pericarditis  
Episthenocardica*)

**Definition** Occlusion of a branch of a coronary artery, with associated cardiac infarction and not infrequently a localised pericarditis

**Etiology** Cardiac infarction is usually due to thrombosis in an atheromatous coronary artery, rarely to syphilis causing a narrowing of the orifice of the coronary arteries, or to an embolus in infective endocarditis

**Pathology** The descending branch of the left coronary artery is most often affected, an infarct forming in the left ventricle Softening, fibrosis or calcification may develop in the infarct There is usually a localised fibrinous pericarditis over the infarct, and aneurysm or rupture of the heart may ensue

**Clinical Findings** The patient is usually a male over the age of 50 He may give a history of previous short attacks of anginal pain In a typical case he is suddenly seized, while at rest, with severe pain in the middle or lower part of the sternum The pain may spread to one or both arms, to the neck, jaw or abdomen. In addition there may be severe dyspnœa, nausea and vomiting The pain is persistent, and lasts for several hours or for a day or so

**On Examination** The patient is usually restless, pale and sweating, and perhaps cyanosed The heart may show no abnormality at the

onset, but the sounds may be distant, or an abnormal rhythm may be present. The pulse: This is often weak, and the rate 90 or 100. The blood pressure characteristically falls, a systolic pressure of 100 mm. Hg, or even 80, being recorded. In some cases there is evidence of venous engorgement, with swelling of the jugular veins, cyanosis, and enlargement of the liver. A few cases have been observed in which there was a complete absence of pain, the patient being suddenly seized with severe dyspnoea. The temperature rises shortly after the onset, and may remain raised for 3 or 4 days; this may be demonstrated in some cases by taking the rectal temperature, which is above normal when the mouth temperature may show no rise. A leucocytosis of about 20,000 per c.mm. is generally present. A pericardial rub often develops in a day or so, serving to confirm the diagnosis. The diagnosis can usually be established by the electrocardiographic findings. In a few cases no changes are present at the onset, but typically, soon after the onset, there is a deviation of the R-T period, usually in opposite directions in leads I and III. Lead IV is of great value in suspected cases of coronary occlusion. In some cases it may show changes, usually elevation or depression of the R-T segment with changes in the T wave, when the three conventional leads are inconclusive. Later, the T wave is inverted in leads I or III ( $T_1$  type or  $T_2$  type), and during convalescence the electrocardiogram is gradually restored to normal.

**Differential Diagnosis.** Coronary obstruction is differentiated from angina pectoris on the following grounds. In coronary obstruction the onset usually occurs when the patient is at rest. He becomes restless, the pain is of long duration, and dyspnoea is often present, with sweating. The pulse is feeble, the temperature rises, the blood pressure falls, the cardiac rhythm may become abnormal and pericarditis ensue, there is a leucocytosis, the electrocardiogram is typical, and the pain is not relieved by nitrites. In angina, on the other hand, the onset is usually related to exercise, the patient stands still, the pain is of brief duration, there is no dyspnoea, and usually little sweating, the pulse is not feeble, the temperature does not rise, the blood pressure rises, the cardiac rhythm is undisturbed, and there is no pericarditis or leucocytosis. The electrocardiographic changes are not so marked, and relief is given by nitrites. An acute abdominal condition may be simulated, when the pain is referred to the abdomen and there is vomiting and collapse. Other causes of severe dyspnoea require exclusion in the painless type of coronary thrombosis. A dissecting aneurysm slowly rupturing into the pericardium may exactly simulate an attack of coronary thrombosis, but no typical electrocardiographic findings are obtained.

**Course and Complications.** The patient may die immediately, or complications, such as congestive heart failure, pericarditis, cerebral thrombosis or embolus, may ensue. Cardiac aneurysm and rupture of the heart are rare complications. Repeated attacks are not infrequent.

**Prognosis.** About half the cases of coronary thrombosis are immediately fatal. About 40% recover from the immediate attack, but death usually occurs within 3 to 5 years. In a small proportion death follows during the course of a week or so from the onset. A second

attack occurs in less than half the cases which survive the first attack. A few recover from the attack, and survive for periods up to twenty years

**Treatment.** Nitrites must not be given. An immediate subcutaneous injection of morphin sulph gr  $\frac{1}{4}$  to  $\frac{1}{2}$  is required, and this is repeated, if necessary to relieve pain, up to gr 1 in 4 hours. If there is congestive heart failure digitalisation should be effected as described on p 217. Oxygen, administered through a nasal catheter or B.L.B. mask (see p 143), may help to relieve pain, restlessness and dyspnoea. A hot water bottle should be placed near the feet. Very little food should be given during the first few days of the illness, and subsequently a diet of about 800 to 1,000 calories is advisable (see p 143). The fluid intake should be restricted to 30 to 40 oz. No aperient should be administered for a week, when an enema may be given if required. The patient should be kept in bed for at least a month from the onset of the illness.

### Angina Pectoris

**Definition.** A condition characterised by paroxysmal attacks of substernal pain, of grave prognosis, and often associated with changes in the aorta, heart or coronary arteries.

**Etiology.** It is now generally believed that in angina the pain impulses arise in the heart muscle as the result of interference with its blood supply. Myocardial ischaemia produces pain, possibly owing to anoxia, possibly as the result of retention of a pain producing factor liberated by muscular contraction and retained locally owing to deficient circulation. The deficient blood supply may be due to disease, obstruction or spasm of the coronary arteries. Further, in some cases anginal symptoms are associated with severe anaemia, diabetes mellitus, hyperthyroidism and hypothyroidism. Angina is not, however, a regular accompaniment of these diseases, there must therefore be some other causative factor than diminished oxygen or sugar supply to the heart muscle, impaired coronary filling or low muscle metabolism. The additional factor in those cases in which angina is present may be coronary narrowing due to lesser degrees of disease or spasm. *Exciting causes* include muscular exercise which is the characteristic instigator of *angina of effort*, and exposure to cold, mental exertion and excitement, a heavy meal and possibly over indulgence in tobacco, which lead to coronary spasm and *spasmodic angina*. Angina is often associated with syphilis, but it may follow an acute illness such as influenza, malaria or rheumatic fever. It tends to run in families and is more common in men, especially after the age of 50. Brain workers are liable to angina.

**Pathology.** The coronary arteries are usually diseased, showing either calcification, endarteritis, or occlusion of their orifice by aortitis or atheroma of the aorta. The aorta is often affected, syphilitic aortitis, atheroma or aneurysm may be present. The myocardium is frequently diseased. If the patient dies in an attack, the heart is usually relaxed and full of blood. Ischaemic fibrosis is often found, resulting from obliterative arteritis of the coronary vessels.



**Clinical Findings.** The patient is usually a man over the age of 50. Angina does rarely affect women, and young people may suffer from it as the result of syphilis or an acute illness, such as influenza. In *angina of effort* the patient complains of attacks of pain which have usually a sudden onset and cessation, and are provoked by exertion, which may, however, only be of a very slight degree, such as walking a few yards, or in more severe cases, turning over in bed or talking. The characteristic feature is that in each case there is a direct and often a quantitative relationship between exercise and the onset and the severity of cardiac pain. Further, with rest the pain is rapidly relieved. Attacks of *spasmodic angina* occur apart from exercise. Further, angina of effort and spasmodic angina may occur simultaneously or consecutively. The attack of *spasmodic angina* does not cease with rest and tends to run its course unless relieved by drugs. An attack of spasmodic angina may be induced by mental excitement, or exposure to cold, such as going into a cold room or between the cold sheets in a bed; a heavy meal is at times provocative. The pain usually begins in the mid line behind the sternum, and may radiate thence to the left shoulder, down the ulnar side of the left arm, to the left side of the neck and jaw, or to the scalp. More rarely the pain radiates to the right side. In some instances the pain is first felt in the region of the xiphisternum or epigastrium (abdominal angina), or in the arteries of the arm. The pain is very severe, continuous while it lasts, and non-throbbing. A sense of constriction, as if the chest were held in a vice, is also felt simultaneously with, or shortly after the pain. This is probably due to contraction of the intercostal muscles. The attack is sometimes accompanied by severe mental anguish and by a sensation of impending death. This is more likely to occur with spasmodic angina. The attack may last for only a second or for several minutes, in some instances a "status anginosus" ensues in which a series of attacks rapidly follow one another. "Angina sine dolore" is also described, in which the patient is suddenly seized by a sensation of imminent death, becomes pale and motionless, and yet experiences no pain. During an attack salivation or vomiting may occur, the attack ceasing with eructation of wind, or a copious flow of urine. Minor attacks vary in severity from slight substernal distress on exertion to definite pain.

**Examination of the Patient During the Attack.** He is usually pale, motionless, and silent; he may, however, be flushed or groan. Frequently the pulse is unaffected, but at times it is feeble. The blood pressure usually rises, and may reach a figure of 340 mm. Hg. syst.; in some instances it is not affected. Sudden death may occur during the attack, perhaps from vagal inhibition or from ventricular fibrillation. The heart may show no abnormality clinically, or there may be enlargement. When the examination is made between the attacks, cardio-vascular degeneration is usually found with arteriosclerosis and increased blood pressure, and shortly after an attack tender spots are often present over the præcordium or along the arm. The Wassermann reaction is positive in a large proportion of cases. An electrocardiogram

taken during or shortly after an attack may show changes comparable with those typical of coronary obstruction (see p 231), but of a lesser degree of intensity. After the attack the electrocardiogram may return to normal.

**Differential Diagnosis** Angina must be differentiated from 1 Pseudo angina (Left inframammary pain Angina innocens) 2 The effort syndrome 3 Coronary thrombosis 4 Cardiac pain from other causes 5 Intercostal pain from other causes 6 Biliary and intestinal colic 7 The scalenus anticus syndrome

1 Pseudo-angina, left inframammary pain or angina innocens. The pain is usually a dull ache, but it may be very severe. It is situated near the apex of the heart and may radiate to the left arm or shoulder. It is present apart from exercise but is frequently accentuated by exercise. The patient is often a woman suffering from a chronic anxiety state. In addition to the pain she may complain of feeling tired, of attacks of sighing respiration, trembling and sweating. No sign of disease of the heart or arteries can be detected. There is usually cutaneous hyperæsthesia over the apex beat or near the inferior angle of the left scapula. The electrocardiogram may show flat  $T_1$  and  $T_2$  waves and inverted  $T_3$  waves. The diagnosis should always be made with caution, as although no cardio vascular disease may be demonstrated clinically, radiologically or electrocardiographically, yet sudden death may occur and post-mortem the coronary arteries are found to be diseased.

2 The effort syndrome and 3 Coronary thrombosis. The differential diagnosis is considered on p 231.

4 Cardiac pain from other causes. Pain resembling that of angina may occur in congestive failure and in acute pericarditis.

5 Intercostal pain from other causes includes a consideration of dorsal spinal arthritis neuralgia myalgia pleurisy, loculated spontaneous left sided pneumothorax and herpes zoster.

6 Biliary and intestinal colic. These conditions should present no difficulties in differential diagnosis if full investigations are carried out.

7 The scalenus anticus syndrome. This is due to pressure of a cervical rib or of the scalenus anticus muscle on the brachial plexus. The pain is neuralgic in character. It may spread from the neck, down the arm to the hand and is increased by rotation of the head to the affected side and by a downward pull on the shoulder. Præcordial pain may also be present.

**Course and Complications** In some cases the patient dies during his first attack of angina, in others the attacks recur with gradually increasing severity and progressive myocardial weakness. Complications include acute oedema of the lungs, cerebral hæmorrhage or thrombosis, coronary thrombosis may follow previous attacks of angina.

**Prognosis** This is always very uncertain, but sudden death occurs in about 60% of cases, and, of the remainder, a few recover whilst others have repeated attacks at intervals up to 20 years before dying either from angina from heart failure or intercurrent disease. Unfavourable

able signs are attacks which are provoked by very slight exertion, attacks which occur during the night, and the presence of pulsus alternans indicating severe myocardial degeneration.

**Treatment. During the Attack:** The immediate indication is to relieve pain. A capsule of amyl nitrite (3 to 5 minims) should be broken and held under the patient's nose for him to inhale. This almost invariably gives relief if the blood pressure is raised. One or two ounces of brandy or whisky frequently relieve the pain, if amyl nitrite is not available. If this fails, a subcutaneous injection of morphin. sulph. gr.  $\frac{1}{2}$  should be given, or an inhalation of chloroform. A subcutaneous injection of atropin. sulph. gr.  $\frac{1}{100}$  should also be given with a view to overcoming vagal inhibition, which may cause death. If there is much flatulence, the patient should be given m. 60 of sal volatile (sp. ammon. aromat. B.P.) with an equal quantity of water. The tenderness over the sternum may be relieved by the application of hot flannels. The patient should retire to bed as soon as possible after the attack has subsided.

**Between the Attacks:** The patient should be warned that the attack indicates severe weakness of the heart and requires rest in bed for 3 to 6 months. During this time he must not smoke, the meals should be small, well masticated and the amount of fluid with meals restricted so as not to cause flatulence. The bowels should be opened regularly every day. No alcohol should be taken. The Wassermann reaction should be determined, and, if positive, a course of treatment with mercury and iodides, and later with intramuscular injections of a bismuth preparation such as Quinostab should be given (see p. 570). If the blood pressure is high, sodium nitrite (gr. 2) may be added to the iodide mixture, or tabella glyceryl. trinitrat. gr.  $\frac{1}{120}$  may be given t.i.d. The tablet should be chewed before swallowing. It has been demonstrated in a series of cases that none of the vaso-dilator drugs is of value in the continuous treatment of angina. For the nervous excitability which is so frequently present, ammon. brom. gr. 10 or Luminal (phenobarbitonum B.P.) gr.  $\frac{1}{2}$  may be given t.i.d., or tab. phenobarbiton. et phenobromin. (B.P.C.), 1 nocte, or a mixture of Nepenthe m. 20 and aspirin gr. 10. A course of insulin, 5 units b.i.d., half an hour before breakfast and dinner, and dextrose 1 oz., taken with these meals, in some cases will diminish the number of attacks or even abolish them. Angina due to anæmia, diabetes mellitus, hyperthyroidism and hypothyroidism, requires treatment of the associated disease together with the administration of sedative or vaso-dilators p.r.n. Subsequently, when the patient is allowed up, the amount of walking exercise may be gradually increased, provided that he feels no distress, his symptoms in this respect being the guiding factor. Running, walking upstairs and climbing slopes should be avoided.

Jonnesco's operation consists in the resection of the whole of the cervical sympathetic and first thoracic ganglion. It is considered dangerous if there is much myocardial degeneration, as the accelerator cardiac nerves are also divided. Total thyroidectomy is sometimes followed by more or less complete relief of cardiac pain. This should

never be performed, until the possibility of masked hypothyroidism (see p 657) has been excluded by BMR determinations. Advanced cardio vascular disease is a contra indication. Promising results have been obtained by the operation of cardio omentopexy. A portion of the omentum is brought through the left diaphragm, and sutured to the edges of the divided pericardium and to the surface of the heart. The blood supply of the ischaemic heart muscle is thus increased.

### Non-penetrating Injuries of the Heart

The heart may be damaged by trauma to the chest wall without the ribs or sternum being fractured. Lesions may occur in the pericardium, myocardium or endocardium. Fibrinous pericarditis, pericardial effusions or adherent pericardium have been noted. The heart may rupture, or an area of scar tissue with subsequent aneurysm of the heart may develop. Injury to the mitral valve has been followed by stenosis. If the blow is severe immediate death may result from ventricular fibrillation. In other cases there is collapse, followed by precordial pain, tightness of the chest, dyspnoea and palpitations. The physical signs vary with the lesion produced. Abnormalities are likely to be present in the electrocardiogram. The T waves may be large or inverted and the R T interval elevated or depressed. Typical coronary T waves may be seen with slurring of the QRS complexes. The patient should be treated as if suffering from coronary occlusion.

## THE ENDOCARDIUM

### Acute Infective Endocarditis

#### *(Malignant or Ulcerative Endocarditis)*

**Definition.** An acute progressive bacterial inflammation of certain parts of the endocardium accompanied by embolic manifestations.

**Etiology.** There is usually an old valvular lesion. A focus of infection is present in some part of the body from which organisms are carried to the heart. The focus is often latent, and may be in the intestines, tonsils, teeth or elsewhere. Malignant endocarditis may also be associated with pneumonia, osteomyelitis, otitis media, typhoid or scarlet fever and diphtheria. More rarely a primary form occurs, in which no extraneous septic focus can be discovered and in which there is no evidence of a previous valvular lesion. The organisms multiply in the heart. They include the *Streptococcus hemolyticus*, the *Diplococcus pneumoniae* (pneumococcus), the staphylococcus or rarely the *Neisseria gonorrhoea* (gonococcus) and the *Haemophilus influenzae*.

**Pathology.** The valves and the endocardium lining the cardiac chambers are chiefly affected. Vegetations form on the surfaces of the valves which are in apposition, such as the auricular aspect of the mitral valve segments and the ventricular surface of the aortic valve cusps. The left side of the heart is usually affected, but in cases of congenital heart disease vegetations form on the right side. They constitute excrescences from the valve or spread along the posterior wall of the left auricle, the wall or septum of the left ventricle (mural

endocarditis) or along the aorta. Deep-seated erosions often occur, and in this way a valve cusp, the interventricular septum or the heart wall may be perforated, or the chordæ tendinæ ruptured. Septic infarcts are liable to form in the spleen, kidneys, brain, intestines, retinae, or, with right-sided lesions, in the lungs.

**Clinical Findings.** The patient may be attacked by malignant endocarditis during the course of some illness, such as pneumonia, puerperal fever, osteomyelitis, otitis media, a cutaneous wound or a boil, or it may develop apparently spontaneously with or without a previous valvular lesion. According to the predominance of certain symptoms and signs several types are described. These are: 1. *The cardiac type.* Here the patient is ill with malaise and a high swinging temperature, the cause of which is uncertain. The pulse is frequent, and the temperature irregular. The heart is usually dilated and varying cardiac murmurs are heard. There is generally progressive anæmia and sweats or rigors ensue. Embolic foci or hæmorrhages are indicated by pain in the region of the spleen, by petechial spots or areas of redness in the skin, by hæmaturia, cerebral symptoms or by diarrhœa. 2. *The septic type.* A primary focus is usually discoverable, such as osteomyelitis. With the onset of endocarditis the patient becomes more gravely ill, variable murmurs are heard at the aortic or mitral areas, and rigors and sweats occur. Embolic phenomena may be detected. 3. *The typhoid type.* The continued fever, drowsiness and mental apathy, with diarrhœa, are suggestive of an enterica group infection. 4. *The cerebral type.* The clinical picture simulates that of meningitis. There is pyrexia with delirium and coma, the cardiac signs often being very slight.

The blood in all types usually shows a progressive leucocytosis, and with counts over 15,000 per c.mm. the blood culture is usually positive. There is also generally a hypochromic anæmia. The urine: Albuminuria, hæmaturia and the causative organisms may be present.

**Course and Complications.** The course is usually rapid, but in the cardiac type it may be prolonged for several weeks. The embolic manifestations mentioned above can be regarded as complications.

**Differential Diagnosis.** The diagnosis often presents difficulties, especially when a case is seen for the first time during the illness, and it is not known whether the cardiac murmur which is heard is of long standing. Malignant endocarditis requires to be differentiated from such conditions as acute simple endocarditis, subacute infective (bacterial) endocarditis, enterica group infections, miliary or acute tuberculosis, septicæmia, malaria, abortus or Malta fever, perinephric abscess, subphrenic or hepatic abscess, meningitis, malignant typhus or small-pox. Acute simple endocarditis: The course of the disease is more benign, emboli are rare and the blood is sterile. Joint symptoms are usually more prominent. Subacute infective endocarditis: The course is often prolonged for 1 or 2 years, and the disease is far less acute. Enterica group infections: The Widal reaction, blood culture and leucopenia serve to differentiate. Miliary or acute tuberculosis: The blood culture is sterile, the heart is unaffected, choroidal tubercles

are often seen, and tubercle bacilli may be found in some cases in the faeces. There is no leucocytosis, and the X ray examination reveals abnormalities in the lung shadows. Septicæmia. The heart is not affected. variable murmurs would not therefore be expected. Malaria. The response to quinine and the presence of parasites in the blood are diagnostic. Abortus or Malta fever. The organisms may be found in the urine and a positive agglutination obtained with the patient's serum. Perinephric abscess. The presence of an infarct in the spleen in endocarditis may cause tenderness in the loin, simulating a perinephric abscess. The cardiac signs are of paramount value in the diagnosis. Meningitis. Examination of the cerebro spinal fluid serves to establish the diagnosis. Malignant typhus or small pox. The rash and course of the illness are diagnostic signs.

**Prognosis.** Death usually occurs in a few weeks.

**Treatment.** The patient must be kept quietly at rest in bed. Various antiseptic substances have been injected intravenously, but any cure by such treatment is improbable. Perchloride of mercury, 0.01 G. in 1 mil. of sterile water may be given every day for 5 doses. Autogenous vaccines and serums are without effect. Sulphanilamide, Sulphapyridine (M. & B. 693) or Sulphathiazole (M. & B. 760), according to the type of organism present in the blood, may be administered for 7 to 10 days in doses of 4 to 6 G., or if carefully controlled by blood counts, for 21 days.

### Subacute Infective Endocarditis

#### (*Endocarditis Lenta*)

**Definition.** A progressive bacterial inflammation of certain parts of the endocardium which tends to run a prolonged course.

**Etiology.** The disease is usually caused by infection with the streptococcus viridans. This is a streptococcus of the salivary or faecal type. **Predisposing causes.** 1 Strain, subacute infective endocarditis was common in soldiers shortly after the 1914-18 war. 2 Age, between 20 and 40. 3 Sex, especially males. 4 Congenital malformation of the valves, particularly bicuspid aortic valves.

**Pathology.** The aortic or mitral valve is usually affected and there may be no evidence of previous valvular trouble. The vegetations are large and greenish in colour, but ulceration of the valves is not common. The vegetations spread to the walls of the heart chambers. The myocardium is often spared.

**Clinical Findings.** The patient is usually a young adult male, who has passed through a period of mental or physical strain. He notices gradually increasing weakness, with dyspnoea on exertion, loss of weight, sweating and feverishness at night. There may also be pains and vague swellings in the joints. In some cases the initial symptom is pain in the spleen, or pleurisy due to a pulmonary infarct in cases associated with congenital cardiac lesions.

**On Examination.** The complexion gradually becomes sallow (*café-au-lait* tinge) and the fingers may be clubbed. Small painful red spots (Osler's nodes) may form and subsequently disappear. They

are seen on the palmar aspects of the tips of the fingers and toes. Petechial hæmorrhages may occur, especially around the neck and shoulders. The temperature is usually about  $99^{\circ}$  to  $100^{\circ}$  F. and runs an irregular course, often being normal for long periods. The pulse is frequent, about 100 to 120. The heart at first often shows no abnormality, later it dilates and a systolic murmur appears at the apex. The appearance of an aortic diastolic murmur is of great significance, indicating endocarditis. The blood: There is a progressive anæmia, with no leucocytosis or a moderate one of about 10,000 per c.mm. The blood culture is usually positive at some period of the disease. The urine: Minute bacterial emboli in the renal glomeruli result in hæmaturia from time to time (focal nephritis, see p. 450). Emboli occur in other sites, such as the spleen, the brain, the retina, the large arteries of the limbs, or the mesentery. The cutaneous lesions mentioned above are probably embolic in nature. An enlarged spleen can often be felt.

**Differential Diagnosis.** It is difficult to make a certain diagnosis in the early stages. Diagnosis is established by the positive blood culture, and by the appearance of cardiac murmurs and of embolic phenomena.

**Course and Complications.** The course is prolonged, the illness often lasting for 1 to 2 years. Complications include the embolic processes described above.

**Prognosis.** This is very grave, but recovery has been recorded; even then a relapse may ensue.

**Treatment.** The patient should be in bed and every measure employed to raise his resistance. Thus fresh air, sunlight or general radiation with ultra-violet light and good food should be employed. Sulphapyridine (M. & B. 693), 4 to 6 G. daily, should be given for 4 weeks. This may be combined with the intravenous administration of heparin during the second and third weeks of treatment. This is not without danger as vascular accidents such as cerebral hæmorrhage may occur. The heparin is given by continuous intravenous drip, 10,000 units being added to every 500 mls of normal saline. The rate of flow is maintained at about 15 to 25 drops a minute, and is regulated to keep the clotting time of venous blood at about 1 hour. The patient should be saturated with ascorbic acid, 200 mg. being given by mouth 4 times a day for 3 days, and subsequently 100 mg. daily. Blood transfusions should be given if there is an anæmia of 3.5 millions red cells per c.mm., or lower. No good results are to be expected from a vaccine or serum, but small blood transfusions of 300 to 500 mls improve the anæmia.

### Acute Simple Endocarditis (Acute Benign Endocarditis)

**Definition.** An acute infective inflammation of certain portions of the endocardium, which pursues a relatively benign course.

**Etiology.** The endocarditis is most commonly associated with rheumatic fever, chorea or scarlet fever. Less frequently it develops

during an attack of tonsillitis, diphtheria, or pneumonia. A recurring endocarditis may appear in cases of long standing valvular lesions.

**Pathology** The lesions probably result from bacteria which are carried to the valves by their blood vessels, or which settle on the valves from the blood in the heart. Vegetations of varying size form on the valves, especially on the auricular surface of the mitral valve and the ventricular surface of the aortic valve, and more rarely on the mural endocardium of the left auricle or ventricle. With congenital heart disease the lesions usually affect the right side of the heart. Emboli may be carried to various sites as in malignant endocarditis (see p 237). There is always an associated carditis.

**Clinical Findings** The patient is usually a young adult between the ages of 10 and 20, who is suffering from one of the acute diseases mentioned above, such as rheumatic fever. The onset of acute endocarditis can only be detected by extremely careful and frequent clinical examinations. Thus the pulse rate may gradually rise, the temperature assume a slightly higher plane and changes appear in the cardiac signs. The apex beat passes out a little to the left, the impulse being more diffuse and forcible, and a murmur appears at the apex or aortic base. The rhythm may also be irregular, due to premature systoles or partial heart block. Rheumatic nodules may be found in various sites (see pp 577-578). The infection may rapidly spread so that the myocardium and pericardium are also involved, a condition of pancarditis. A typical case presents the following picture. The patient lies prostrate in bed, with rapid shallow respirations of about 40. The lips and ears are cyanosed and the slightest movement of the hands or arms intensifies the dyspnoea. The pulse may be slow, 40 to 60, and irregular owing to "coupled beats." The heart is enlarged to the left, a systolic and diastolic murmur are present at the apex, the sounds are weak, and a localised area of pericardial friction may be heard over the pulmonary base.

**Differential Diagnosis** In the early stages there is the greatest difficulty in distinguishing a mitral murmur due to endocarditis from that produced by dilatation of the valve ring. The diagnostic points are discussed on p 242.

**Course and Complications** The valvular lesion is often progressive. Thus definite signs of mitral stenosis may appear in a few months. Complications include pericarditis or pleurisy, and in some cases malignant endocarditis may supervene.

**Prognosis** There is likely to be permanent valvular damage, either slight and non progressive, or severe with an increasing valvular defect. Adherent pericardium may still further embarrass the heart's action.

**Treatment.** The patient must be kept absolutely flat at rest in bed and not allowed to do anything for himself, until the pulse, temperature and respirations are normal. He may then be taken off "absolute" rest, but must lie flat in bed for another month, then he is gradually raised in bed with pillows. A careful watch must be kept upon the size of the heart and the heart sounds during convalescence. After



2 to 6 months in bed he is allowed to lie on a couch for graduated periods, if satisfactory progress is being made. He should not walk for at least 8 months, and then only slowly. No running is permissible for 2 years. No special drugs are required—salicylates should be discontinued if a cardiac depressant effect is observed, as shown by weakening of the pulse or dilatation of the heart. Tincture of digitalis in doses of m. 10 to 15 t.i.d. should be given if the pulse is rapid. If there is heart-block or a slow rhythm associated with premature systoles and coupled beats, no digitalis should be given. Stimulants, such as Coramine (nikethamidum B.P. Add.) 1·5 mil. or Cardiazol (leptazolum B.P. Add.) 1 mil. should then be given subcutaneously every 6 hours. If there is any septic focus, as in the tonsils, it should be eradicated in the later stages of convalescence.

### Chronic Endocarditis

#### *(Chronic Valvular Disease of the Heart)*

**Definition.** Inflammatory and degenerative changes in the valves of the heart.

**Etiology.** The inflammatory changes are often sequelæ of acute endocarditis occurring in association with acute rheumatism, scarlet fever, influenza or diphtheria. The degenerative lesions may be associated with syphilis, arteriosclerosis, high blood pressure and chronic toxæmia.

**Pathology.** Deformity of the valve cusps or ring is likely to occur, but the mural endocardium remains practically unaffected. The mitral and aortic valves are the usual seats of the trouble.

### Mitral Regurgitation

#### *(Mitral Incompetence)*

**Definition.** Reflux of blood from the left ventricle to the left auricle during the ventricular systole.

**Etiology.** The regurgitation may be due to dilatation or thickening of the valve ring, or to alterations in the valve cusps or chordæ tendineæ preventing effective closure. The valve ring: The orifice may dilate in association with myocardial weakness and dilatation of the left ventricle, in febrile or anæmic states, or in association with enlargement of the left ventricle in aortic disease, or increased blood pressure. It may be thickened from organic changes associated with previous rheumatic or other infections, or with arteriosclerosis. The valve cusps and chordæ tendineæ may be deformed, thickened and adherent as the result of inflammatory changes, and in some cases rupture may occur.

**Pathology.** In addition to the changes in the ring or valve cusps described above, there is often in valvular cases a certain degree of narrowing of the valve orifice (stenosis) and some enlargement of the left ventricle, and later of the left auricle, and finally of the right side of the heart.

**Clinical Findings.** The patient often gives a history of previous rheumatic infection. He usually does not complain of any symptoms

during an attack of tonsillitis, diphtheria, or pneumonia. A recurring endocarditis may appear in cases of long standing valvular lesions.

**Pathology** The lesions probably result from bacteria which are carried to the valves by their blood vessels, or which settle on the valves from the blood in the heart. Vegetations of varying size form on the valves, especially on the auricular surface of the mitral valve and the ventricular surface of the aortic valve, and more rarely on the mural endocardium of the left auricle or ventricle. With congenital heart disease the lesions usually affect the right side of the heart. Emboli may be carried to various sites as in malignant endocarditis (see p. 237). There is always an associated carditis.

**Clinical Findings** The patient is usually a young adult between the ages of 10 and 20, who is suffering from one of the acute diseases mentioned above, such as rheumatic fever. The onset of acute endocarditis can only be detected by extremely careful and frequent clinical examinations. Thus the pulse rate may gradually rise, the temperature assume a slightly higher plane and changes appear in the cardiac signs. The apex beat passes out a little to the left, the impulse being more diffuse and forcible, and a murmur appears at the apex or aortic base. The rhythm may also be irregular, due to premature systoles or partial heart block. Rheumatic nodules may be found in various sites (see pp. 577, 578). The infection may rapidly spread so that the myocardium and pericardium are also involved, a condition of pancarditis. A typical case presents the following picture. The patient lies prostrate in bed, with rapid shallow respirations of about 40. The lips and ears are cyanosed and the slightest movement of the hands or arms intensifies the dyspnoea. The pulse may be slow, 40 to 60, and irregular owing to "coupled beats." The heart is enlarged to the left, a systolic and diastolic murmur are present at the apex, the sounds are weak, and a localised area of pericardial friction may be heard over the pulmonary base.

**Differential Diagnosis** In the early stages there is the greatest difficulty in distinguishing a mitral murmur due to endocarditis from that produced by dilatation of the valve ring. The diagnostic points are discussed on p. 242.

**Course and Complications** The valvular lesion is often progressive. Thus definite signs of mitral stenosis may appear in a few months. Complications include pericarditis or pleurisy, and in some cases malignant endocarditis may supervene.

**Prognosis** There is likely to be permanent valvular damage, either slight and non progressive, or severe with an increasing valvular defect. Adherent pericardium may still further embarrass the heart's action.

**Treatment.** The patient must be kept absolutely flat at rest in bed and not allowed to do anything for himself, until the pulse, temperature and respirations are normal. He may then be taken off "absolute" rest, but must lie flat in bed for another month, then he is gradually raised in bed with pillows. A careful watch must be kept upon the size of the heart and the heart sounds during convalescence. After

2 to 6 months in bed he is allowed to lie on a couch for graduated periods, if satisfactory progress is being made. He should not walk for at least 8 months, and then only slowly. No running is permissible for 2 years. No special drugs are required—salicylates should be discontinued if a cardiac depressant effect is observed, as shown by weakening of the pulse or dilatation of the heart. Tincture of digitalis in doses of m. 10 to 15 t.i.d. should be given if the pulse is rapid. If there is heart-block or a slow rhythm associated with premature systoles and coupled beats, no digitalis should be given. Stimulants, such as Coramine (nikethamidum B.P. Add.) 1·5 mil. or Cardiazol (leptazolium B.P. Add.) 1 mil. should then be given subcutaneously every 6 hours. If there is any septic focus, as in the tonsils, it should be eradicated in the later stages of convalescence.

### Chronic Endocarditis

#### *(Chronic Valvular Disease of the Heart)*

**Definition.** Inflammatory and degenerative changes in the valves of the heart.

**Etiology.** The inflammatory changes are often sequelæ of acute endocarditis occurring in association with acute rheumatism, scarlet fever, influenza or diphtheria. The degenerative lesions may be associated with syphilis, arteriosclerosis, high blood pressure and chronic toxæmia.

**Pathology.** Deformity of the valve cusps or ring is likely to occur, but the mural endocardium remains practically unaffected. The mitral and aortic valves are the usual seats of the trouble.

### Mitral Regurgitation

#### *(Mitral Incompetence)*

**Definition.** Reflux of blood from the left ventricle to the left auricle during the ventricular systole.

**Etiology.** The regurgitation may be due to dilatation or thickening of the valve ring, or to alterations in the valve cusps or chordæ tendineæ preventing effective closure. The valve ring: The orifice may dilate in association with myocardial weakness and dilatation of the left ventricle, in febrile or anæmic states, or in association with enlargement of the left ventricle in aortic disease, or increased blood pressure. It may be thickened from organic changes associated with previous rheumatic or other infections, or with arteriosclerosis. The valve cusps and chordæ tendineæ may be deformed, thickened and adherent as the result of inflammatory changes, and in some cases rupture may occur.

**Pathology.** In addition to the changes in the ring or valve cusps described above, there is often in valvular cases a certain degree of narrowing of the valve orifice (stenosis) and some enlargement of the left ventricle, and later of the left auricle, and finally of the right side of the heart.

**Clinical Findings.** The patient often gives a history of previous rheumatic infection. He usually does not complain of any symptoms

unless the heart muscle itself begins to fail (failure of compensation). He will then notice a train of symptoms such as undue dyspnoea on exertion, palpitations, and later swelling of the ankles, cough and expectoration.

*On Examination* In a well marked case of fully compensated mitral regurgitation the only abnormal signs will be in the heart. *Inspection* The apex beat may be displaced a little in the fifth and sixth spaces, about 4 inches from the mid line. *Palpation* The impulse is forcible, and a systolic thrill is at times felt at the apex. *Percussion* The area of cardiac dulness is increased a little downwards and to the left. *Auscultation* Both sounds are heard at the apex and base. At the apex there is a systolic murmur accompanying the first sound, and in some cases apparently replacing it, although graphically the first sound is always present. The murmur may be soft or loud, and is conducted outwards towards the axilla in some cases being heard as far round as the angle of the scapula or even the spine. The maximum intensity is over the apex. With failure of compensation other signs appear. There is some cyanosis of the face and dyspnoea on slight exertion. The apex of the heart may pass out further to the left and the rate increase. Irregularity may be noted, due to premature systoles or auricular fibrillation. Further signs of venous engorgement may be found, such as enlargement of the jugular veins in the neck, dilatation of the tricuspid valve, as indicated by a systolic murmur over the tricuspid valve area. The liver may be enlarged and pulsating, and rales may be heard at the bases of the lungs. (Edema of varying degree may be seen in the ankles and legs, and ascites may be present. The urine is often diminished and contains albumin or blood.

*Differential Diagnosis* The characteristic sign of mitral regurgitation is the apical systolic murmur which is conducted outwards and rarely there is a systolic thrill. *The conditions under which a systolic murmur may be heard at the apex of the heart are as follows* —

1 *Physiological* In an apparently normal individual a soft systolic murmur may be heard at the apex of the heart. This is due to a temporary dilatation of the valve ring, and although it may produce no apparent disturbance of function, it is not a normal condition.

2 *Intracardiac* The murmur may be (a) *Hæmic* Associated with anæmia and dilatation of the valve ring and often heard better at the base than at the apex of the heart. It is soft and often varies with position, rest, exercise and respiration. (b) *Fébrile* This is a soft and localised apical murmur, which may occur during fevers without any evidence of dilatation of the heart, but is probably due to dilatation of the valve ring. (c) *Due to dilatation of the mitral valve ring (relative incompetence)*, as in a dilated and hypertrophied heart. This is a variety of mitral regurgitation. (d) *Organic* Due to changes in the valve ring or segments as described above. These murmurs are often musical and conducted towards the axilla. The murmur is little affected by change of position, exercise or by respiration. There is often cardiac enlargement. An aortic systolic murmur may be transmitted to the apex.

3. *Exocardial.* A cardio-respiratory systolic apical murmur may be due to pleuro-pericardial adhesions, whereby the pressure of air in an adjacent portion of lung is affected by the heart beat. The murmur is usually late systolic and is affected by the phases of respiration. In overacting hearts the ventricular contraction causes the adjacent portion of lung to expand rapidly and suck in air, producing a cardio-respiratory short and blowing murmur. A systolic murmur may be heard in acute pericarditis, but more often there is a "to and fro" murmur.

*Prognosis.* The prognosis in mitral regurgitation depends upon the condition of the myocardium, arteries and kidneys, and is adversely affected by the presence of mitral stenosis. In a simple case, with a healthy myocardium, the heart being normal in size, the exercise tolerance good, and there being no evidence of infection, a full and active life may be expected for many years.

*Treatment.* No treatment is required unless there are indications of failure of compensation. The patient must then be instructed to live well within his reserve of cardiac power and to avoid all strenuous exercise. If decompensation occurs the treatment is as described on p. 227 for the failing heart.

### Mitral Stenosis

*Definition.* Narrowing of the mitral valve.

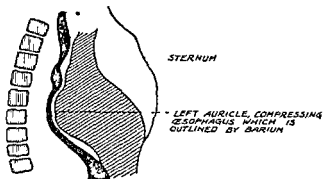
*Etiology.* Mitral stenosis is usually inflammatory in origin, being a sequel of acute rheumatic endocarditis in about 90% of cases, but it also follows other infections, such as scarlet fever, diphtheria or influenza. Signs of stenosis do not show themselves for several months or years after an acute rheumatic attack. Less frequently mitral stenosis is an atheromatous lesion, occurring in people over middle age, and associated with chronic nephritis. Sex: Females predominate.

*Pathology.* The valve ring may be narrow, sclerotic and slit-like (*Corrigan's button-hole stenosis*), or the cusps may be adherent and the chordæ tendinæ shortened (*funnel-shaped stenosis*). The former is more common in adults, the latter in children. Secondary changes appear in the heart and elsewhere, such as dilatation and hypertrophy of the left auricle and right ventricle, congestion of the lungs and liver, etc.

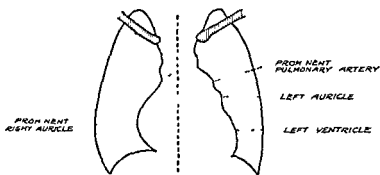
*Clinical Findings.* In a typical case, in which symptoms of failure of compensation are not marked, the patient is often a young adult who gives a history of an infection, particularly that of rheumatic fever, some years before. In many cases, however, no such history can be obtained. The patient may notice no symptoms, or he may complain of palpitations, dyspnoea on exertion, or slight swelling of the ankles towards the evening.

*On Examination:* The mitral facies may be noted, in which the cheeks and lips have a high colour, with slight cyanosis of the face and ears. The heart. Inspection: The apex beat is usually visible in its normal site. Palpation: The impulse is short, forcible or slapping, at the apex. A presystolic apical thrill may be felt. This is a rough

vibrating sensation, comparable with that experienced when the hand is placed on the back of a purring cat. It can be tuned by simultaneously feeling the carotid impulse. Percussion The right border of the cardiac dulness may be a little "out." Auscultation: The first sound at the apex is usually accentuated, and the second sound rather weak. The pulmonary second sound is generally accentuated. The various stages of mitral stenosis are indicated by the sounds heard at the apex. The duration and intensity of the diastolic murmur depend



(a) *Right Anterior Oblique or Oblique I* (The patient is turned so that the right shoulder rotates forward through 45 degrees)



(b) *A-P position*

FIG 12 DIAGRAM OF RADIOGRAPHIC APPEARANCES OF THE HEART IN MITRAL STENOSIS.

upon the degree of stenosis and the rapidity and force of the heart beat. With advanced stenosis the diastolic murmur may be very faint if the auricular contraction is feeble and the auricle is obstructed by clots *Stage I.* A short presystolic murmur is only heard after the patient has exercised, as by sitting up and lying down 20 times, the apex region being then auscultated with the patient lying on the left side. Inhalation of amyl nitrite m. 5, may also unmask the murmur. The presystolic murmur is usually very localised to a point just internal to the apex beat. *Stage II.* A definite rough, rasping, low-pitched, crescendo presystolic murmur ending suddenly in an accentuated first sound, is heard at the apex, apart from exercise. It is usually intensi-

fled when the patient lies on his left side. The pulmonary second sound may be reduplicated. *Stage III.* A mid-diastolic and presystolic murmur, and perhaps an early diastolic murmur, are audible at the apex. The stenosis is more marked, the early diastolic murmur being produced by blood flowing from the left auricle to the left ventricle, before auricular systole begins. In addition to these diastolic murmurs a systolic murmur is also often heard at the apex, indicating that the mitral valve is incompetent. The second sound may disappear at the apex. *Stage IV.* The typical irregularity of auricular fibrillation is now noted and the presystolic murmur disappears if the rate of heart beat is slow; further, when the ventricle is beating slowly the early diastolic murmur becomes softer, although the mid- and early-diastolic murmurs persist. If the rate is rapid the murmur still occupies the whole of diastole. With fibrillation, signs of failure of compensation (see p. 225) are often present. The pulse is usually of low systolic tension, but the diastolic reading may be raised.

*Radiographic Findings:* An antero-posterior X-ray in a developed case shows prominence of the pulmonary artery, conus arteriosus and left auricular appendix. The pulmonary artery becomes prominent owing to hypertrophy of the right ventricle. In the right anterior oblique position, in which the patient is rotated with the right shoulder forwards, through 45°, a shadow in the retrocardiac space due to the dilated left auricle may be seen. With a barium swallow the œsophagus may be seen curving round the dilated left auricle (see Fig. 12). The electrocardiogram is likely to show right axis deviation, with a big or bifid P<sub>1</sub> wave.

*Differential Diagnosis.* Accurate timing of apical murmurs is essential. When this is done difficulty usually only occurs in the early stages, when there may be doubt between a short presystolic murmur heard after exercise and an accentuated or reduplicated first sound at the apex. Undoubtedly many cases have been diagnosed as early mitral stenosis, when the subsequent course has shown that no stenosis is present. With aortic regurgitation a diastolic murmur (Austin Flint murmur, see p. 246) may be heard at the apex. In adherent pericardium a presystolic murmur may rarely be heard at the apex, although there is no mitral stenosis: this is due to dilatation of the valve ring and is associated with a systolic apical murmur.

*Course and Complications.* Mitral stenosis usually pursues a prolonged course, and auricular fibrillation and heart failure do not necessarily ensue. Complications include: Embolism, a clot may be detached from the left auricle and settle in the brain, spleen or kidneys, etc.; or from the right auricle and cause pulmonary embolus with hæmoptysis. Congestion of the lungs and bronchitis. Premature systoles, auricular fibrillation and heart failure. Auricular fibrillation, this is often of short duration at the onset, but later it tends to be permanent. Each attack of heart failure is more serious. Laryngeal paralysis, from pressure of the dilated left auricle on the left recurrent laryngeal nerve. Recurrent attacks of endocarditis may occur.

*Prognosis.* The presence of mitral stenosis usually means that the

patient's life is limited both in activity and in duration. It is a more serious lesion than mitral regurgitation. It may cause sudden death, usually from cerebral embolus.

**Treatment.** The patient must be instructed to live well within his reserve of cardiac power. Failure of compensation and auricular fibrillation are treated as described on pp. 217, 227.

### Aortic Regurgitation

**Definition.** Reflux of blood through the aortic valve.

**Etiology.** Five types are described. 1 Arteriosclerotic. A degenerative lesion, usually associated with syphilis. This accounts for 30% to 40% of all cases. 2 Endocarditic. This is met with in young people, especially as a complication or sequela of acute rheumatism. 3 Traumatic. A valve cusp may rupture from sudden strain, if it is previously diseased by syphilis or subacute infective endocarditis. 4 Congenital. Two valve cusps may be fused, so that they do not close properly. 5 Relative incompetence. Dilatation of the first part of the aorta and of the aortic ring may prevent closure of the cusps.

**Pathology.** The aortic valve cusps may be puckered and shrunken, with associated stenosis, or show vegetations and destructive lesions due to endocarditis. There is usually hypertrophy of the left ventricle, the heart being enlarged (*cor bovinum*). There may be fibroid myocardial changes, and atheroma of the aorta and coronary arteries.

**Clinical Findings.** The patient, who is usually an adult male, may give a history of an attack of rheumatic fever some years ago, or of having contracted syphilis earlier in his life. He may complain of giddiness or faintness at times, or of headache, palpitations and dyspnoea on exertion. In some cases præcordial pain is the first symptom noticed. At times the onset is sudden with fainting or severe dyspnoea.

**On Examination.** Facial pallor is a characteristic feature, although some authorities consider it is only a sign of bacterial endocarditis. The patient may have an anxious expression, and pulsation of the carotid arteries may be noticeable. **The heart.** **Inspection.** The cardiac impulse is forcible and diffuse, and the apex beat is in the sixth or seventh left space, external to the nipple line. **Pulsation.** may be seen in the suprasternal notch. **Palpation.** The impulse is forcible and usually displaced downwards and outwards. There is generally no thrill. Rarely a diastolic thrill is felt at the aortic base. **Per-cussion.** The area of cardiac dulness is increased downwards and to the left. **Auscultation.** Both sounds are usually heard at the apex. There may be a systolic murmur at the apex due to mitral regurgitation. An Austin Flint murmur may be heard at the apex. This is a rumbling, long diastolic murmur, which is thought to be due to the pressure of regurgitant blood in the aorta on the anterior cusp of the mitral valve. At the aortic base, or at times at the pulmonary base, the second sound is replaced by a diastolic murmur, or the second sound may be heard accompanied by a murmur. The diastolic murmur is soft or rough. When heard at the aortic base it is usually conducted down to the



xiphisternum, and more rarely up into the neck. When present over the pulmonary base it may be conducted downwards along the left border of the sternum towards the apex. This is believed by some to indicate a lesion of the left posterior cusp of the aortic valve. The first sound at the aortic area may be normal, or accompanied or replaced by a systolic murmur if there is roughening or stenosis of the aortic valve. The rhythm is usually regular, and the rate may be normal or increased. The arteries are often thickened. The pulse: This is typically of the collapsing type, and it is known as the water-hammer or Corrigan's pulse. The character of the pulse is best experienced if it is felt with the middle phalanges of the fingers rather than with the tips, the patient's arm being first at a low level and then elevated. In the latter position a short sharp tap is felt with systole and the characteristic collapsing sensation with each diastole. The blood pressure: The systolic pressure is high and the diastolic pressure low, such as 170/60 mm. Hg., there being a high pulse pressure. The blood pressure in the legs is higher than that in the arms; this is due to a compensatory mechanism to maintain the cerebral circulation. On auscultation over the femoral artery a diastolic murmur may be heard (Duroziez' murmur). Capillary pulsation (due chiefly to vaso-dilatation): On compressing the tip of the nail, so as partially to blanch the nail bed, pulsation may be seen at the junction of the white and red areas; similarly by drawing the finger nail along the forehead and causing a red line to form, capillary pulsation may be seen at the edges of the line. Capillary pulsation may be seen inside the lips, when they are compressed with a glass slide, or in the retinal vessels with the aid of the ophthalmoscope.

**Differential Diagnosis.** An aortic diastolic murmur may be very difficult to hear in the early stages, absolute silence in the room being essential. The patient should be examined sitting well forward, erect and lying, with the breath held in full inspiration and in full expiration, both before and after exercise, and auscultation should be practised along the right and left borders of the sternum. In a well-developed case the murmur is one of the easiest to detect. There are then usually the concomitant signs of aortic reflux mentioned above.

**Course and Complications.** Aortic regurgitation often pursues a prolonged course, with little effect upon function, so that for instance the patient can play a hard game of tennis. Failure to maintain the cerebral circulation causes giddiness or faintness. Recurrences of endocarditis will result in fever and possibly in embolism. Extension of atheromatous changes may cause angina or aneurysm. Heart failure, with normal or abnormal rhythm, results, from myocardial weakness. Aortic stenosis and mitral regurgitation place additional strain upon the myocardium, but a combination of aortic regurgitation and mitral stenosis did not prevent a man from being a successful marathon runner. Hemiplegia may result from cerebral hæmorrhage.

**Prognosis.** In traumatic cases, sudden or rapid death is the rule. With recurrent endocarditis (malignant endocarditis or subacute infective endocarditis), death usually occurs in a few months or in a year or so. The most favourable outlook is in cases due to a previous

endocarditis which is completely arrested. With degenerative lesions coronary disease and myocardial failure usually eventually ensue. When heart failure occurs the prognosis is more unfavourable than is the case with mitral lesions. In any case the possibility of sudden death must be remembered.

**Treatment.** The Wassermann reaction should be determined, and, if positive a course of anti syphilitic treatment given, with Pot iod gr 5 to 30 liq hydrarg perchlor m 20 to 60 sp chlorof m 7, aquamad fl oz 1 Fl oz 1 t.d.s. p.c., for 2 to 3 months (see also p 259). This is followed by weekly intramuscular injections of 0.3 G Quinostab (quinine iodobismuthate) for 12 doses. Small doses of neoarsphenamine are then given intravenously, starting with 0.1 G and increasing to 0.3 G for 6 doses. Preparations of arsenic, such as neoarsphenamine, should only be used with great caution after iodide administration, as a local reaction with swelling of the orifices of the coronary arteries may cause sudden death. They should never be employed in cardiovascular syphilis in which there is angina. A second course of pot. iod and mercury is then given for 2 to 3 months, followed, if the Wassermann reaction remains positive by another course of Quinostab and neoarsphenamine. Anæmia may be helped by a mixture containing Ferri et ammon cit gr 15 to 20 sod bicarb gr 10, sp chlorof m. 7, infus gent co rec ad fl. oz 1 Fl oz 1 t.d.s p.c. For heart failure or auricular fibrillation digitalis should be administered as described on p 217. Pain is an indication for more complete rest.

### Aortic Stenosis

**Definition.** Narrowing of the orifice of the aortic valve.

**Etiology.** Aortic stenosis usually results from inflammatory changes following rheumatic endocarditis or, in elderly people, it may be caused by calcareous degeneration in the cusps. Rarely, if ever is it due to syphilis. Very rarely it is congenital.

**Pathology.** The valve cusps are thickened, shrunken and adherent. They lose their mobility and the lumen of the valve orifice is narrowed. Calcareous particles may be found in the cusps. The valve cusps may be judged post-mortem to be incompetent, although no regurgitant murmur was detected during life. The left ventricle is usually hypertrophied and mitral incompetence is often present. In relative aortic stenosis the orifice is normal in size, but the aorta beyond is dilated.

**Clinical Findings.** The patient is generally a male over middle age. A history of rheumatic fever and rarely of syphilis, may be obtained. He may complain of præcordial pain or distress on exertion of syncopal attacks due to exaggeration of the carotid sinus reflex, or of symptoms due to some complication such as a retinal venous thrombosis disturbing vision.

**On Examination.** The patient often appears healthy. The heart. **Inspection.** The cardiac impulse is forcible, and the apex beat is seen in the fifth or sixth left space a little external to the nipple line.

**Palpation:** A rough systolic thrill is felt over the aortic base. The apex beat is forcible. **Percussion:** The area of cardiac dulness is increased, especially downwards and to the left. **Auscultation:** At the apex the first sound may be replaced or accompanied by a systolic murmur, due to mitral regurgitation. The second sound at the apex is weak. Over the aortic base a rough systolic murmur is heard, conducted upwards into the neck on the right side, and also downwards over the sternum. The murmur, however, becomes weaker before the point is reached at which the apical systolic murmur is picked up. The aortic second sound is usually weak, and may be absent. An aortic diastolic murmur may be heard, indicative of aortic reflux (double aortic disease). The pulmonary second sound is often weak, and may be practically inaudible if tricuspid regurgitation is also present, so that it is difficult to hear a second sound at any point over the heart. The aortic systolic murmur may at times be heard at the back, the maximum intensity being just to the left of the fourth thoracic vertebra. The rate of cardiac beat is often slow, such as 50 to 60, and the rhythm regular. **The arteries:** The radial or brachial arteries are often thickened. **The pulse:** This presents the slow, small, but sustained type of impulse, the artery remaining filled between the beats. A tracing shows the anacrotic type, the dicrotic wave being absent or poorly marked. **The blood pressure:** The systolic pressure is not raised, but the diastolic reading may be high, such as 140/100.

**Differential Diagnosis.** Aortic stenosis is a rare disease, and must not be diagnosed solely on the presence of an aortic systolic murmur. This may only indicate roughening of one of the valve cusps, atheroma of the aorta, aneurysm, overaction of the left ventricle, or anæmia. The diagnostic features are the systolic thrill, the systolic murmur propagated to the neck, and the small anacrotic pulse. If the aortic second sound is absent, there is probably extensive valvular disease.

**Course and Complications.** The lesion is usually slowly progressive. Complications include myocardial degeneration, thrombosis of retinal vessels, and cerebral thrombosis or hæmorrhage.

**Prognosis.** Death usually occurs within a few years of diagnosis in degenerative cases. *The outlook is more favourable if the lesion is of the inflammatory type.*

**Treatment.** The Wassermann reaction should be determined, and, if positive, a course of anti-syphilitic treatment should be given, as described on p. 248. In all cases strain and over-exertion must be avoided.

### Tricuspid Regurgitation

**Definition.** Regurgitation of blood from the right ventricle to the right auricle.

**Etiology.** Tricuspid regurgitation is usually associated with dilatation of the right ventricle, and secondary to valvular lesions in the left side of the heart. It may occur with pulmonary fibrosis, chronic bronchitis and emphysema (cor pulmonale, see p. 223). Endo-

carditis causing incompetence rarely affects the tricuspid valve. A congenital variety may also be met with.

**Clinical Findings** The onset may be comparatively sudden with acute right sided heart failure (see p 226).

**On Examination** The face is cyanosed. Venous engorgement may be apparent in the neck, the jugular veins being distended and pulsating with each ventricular systole. The jugulars, when emptied by the finger will be seen to fill from below. The liver may be enlarged, tender and on bimanual palpation systolic expansion may be felt. The heart. **Palpation** It is uncommon to feel a systolic thrill over the tricuspid area. **Percussion** The heart is enlarged to the right. **Auscultation** There is a soft systolic murmur, with its maximum intensity over the fourth right costal cartilage and lower part of the sternum. It may be conducted a little towards the right nipple, or heard at the back near the angle of the right scapula. The pulmonary second sound is faint. Other murmurs due to lesions of the mitral or aortic valves may be present. There are usually râles at the bases of the lungs and ascites may be present.

**Differential Diagnosis** The systolic murmur must be differentiated from that due to mitral regurgitation. The characteristic features of tricuspid regurgitation have been detailed above.

**Course and Complications** Relative incompetence may disappear with adequate treatment.

**Prognosis** This is serious, as the lesion is indicative of a severe degree of heart failure.

**Treatment.** This is as described on p 227. Venesection and digitalisation are usually required.

### Tricuspid Stenosis

**Definition** Narrowing of the orifice of the tricuspid valve.

**Etiology** Tricuspid stenosis is a very rare lesion, either resulting from previous endocarditis or being congenital in origin.

**Pathology** The narrowed tricuspid orifice is often associated with mitral stenosis.

**Clinical Findings** The patient is usually cyanosed and may be drowsy and cold. The heart. **Palpation** A presystolic thrill may be felt over the right side of the lower part of the sternum. **Percussion** The heart is enlarged to the right. **Auscultation** A diastolic murmur, usually mid-diastolic, is heard with maximum intensity over the lower part of the sternum conducted slightly upwards and to the right. The liver. It may be possible to detect presystolic (auricular systolic) pulsation. The liver is usually enlarged. There is frequently œdema of the ankles and ascites develops later.

**Differential Diagnosis** As tricuspid stenosis is so rare, it must be very carefully differentiated from mitral stenosis with which it is usually associated. With the onset of auricular fibrillation the diastolic murmur usually disappears.

**Treatment.** This is as for auricular fibrillation or for right sided cardiac failure (see pp 217, 227).

### Pulmonary Regurgitation

**Definition.** Regurgitation of blood through the pulmonary valve.

**Etiology.** Pulmonary regurgitation may occur as a complication of mitral stenosis, due to increased pressure in the pulmonary circuit with dilatation of the pulmonary artery. It may also be due to infective endocarditis grafted on a congenital pulmonary stenosis, or more rarely it occurs as a congenital lesion combined with pulmonary stenosis. It is a very rare valvular lesion.

**Clinical Findings.** The characteristic sign is a diminuendo soft diastolic murmur, following the second sound, with maximum intensity in the second and third left spaces, near to the sternum. It is conducted down the left border of the sternum. The pulmonary diastolic murmur, which may be heard in advanced cases of mitral stenosis, is known as a Graham Steell murmur, and is thought to be due to pulmonary regurgitation.

**Differential Diagnosis.** It is very difficult to diagnose pulmonary regurgitation from early cases of aortic reflux, in which the pulse is not collapsing and other signs of aortic disease are absent.

### Pulmonary Stenosis

**Definition.** Narrowing of the pulmonary valve.

**Etiology.** Pulmonary stenosis is usually a congenital lesion, and is described on p. 252. It may be due to compression of the pulmonary artery by a mediastinal tumour or an aortic aneurysm. Rarely it is caused by infective endocarditis.

## CONGENITAL DISEASE OF THE HEART

**Etiology.** Congenital heart disease is usually caused by developmental errors, more rarely by foetal endocarditis. The right side of the heart is generally affected, probably owing to the higher pressure which obtains there during foetal life. It is more common in boys, particularly in first-born children.

**Pathology.** Congenital lesions may be classified as:—

1. *Abnormalities of Position.* The heart may be situated external to the chest wall, in the neck or in the abdomen (ectopia cordis), or it may be on the right side of the body (dextrocardia) with transposition of the aorta and pulmonary artery. In the latter case the other viscera may be in their normal site or transposed.

2. *Septal and Fetal Passage Defects.* (a) The interauricular or interventricular septum may be absent (cor triloculare). (b) The interauricular and interventricular septa may be absent (cor biloculare). (c) The interauricular septum may be imperfect or a patent foramen ovale may be present. (d) The interventricular septum may be incomplete, usually at the site of the pars membranacea, near the upper end of the septum. (e) The ductus arteriosus may remain patent. Normally this closes by the eighth day after birth, and if it remains patent it does so as a mechanism compensatory to other defects. These include pulmonary stenosis and aortic stenosis, the patent ductus

allowing blood to pass to the pulmonary artery from the aorta in the former case, and to the aorta from the pulmonary artery in the latter. A patent interventricular septum is also often present.

3. *Defects of the Main Vessels.* (a) Pulmonary stenosis. This may imply actual atresia of the pulmonary artery, or narrowing of the artery at the valve level or at the infundibulum. (b) Aortic stenosis. The occlusion may occur at the valve ring, between this and the entrance of the ductus arteriosus, or at the point just below the entrance of the ductus arteriosus (coarctation of the aorta, see p. 253).

4. *Valvular Defects.* (a) The pulmonary valve. Pulmonary stenosis may be due to shrinkage of the valve cusps, or to narrowing of the valve ring, or a supernumerary cusp may be present often associated with a patent foramen ovale or persistent ductus arteriosus. Only two cusps may be present, or there may be a supernumerary one. Pulmonary regurgitation is uncommon. (b) The tricuspid valve. Congenital regurgitation or stenosis may occur. (c) The aortic valve. Two cusps only may be present, and the valve be incompetent, or there may be a supernumerary cusp. (d) The mitral valve. This is rarely stenosed.

5. *Defects of Conducting Tissue.* Congenital heart-block is usually due to a deficiency of the upper part of the interventricular septum, the auriculo-ventricular bundle ending in a band of fibrous tissue.

6. *Combined Lesions*, such as Fallot's tetralogy. In this there is stenosis or hypoplasia of the pulmonary artery, a defect in the interventricular septum, the aorta communicates with both ventricles, and the right ventricle is hypertrophied.

*Clinical Findings.* In a typical case, in which there is deficient aeration, the condition is noted at or shortly after birth. The baby is cyanosed and blue (*morbus caeruleus*), and a murmur is audible over the præcordium. Later in life, if the child is examined by the doctor for the first time, a history may be obtained that he has been blue from birth, and the parents may have been told that the heart was affected. There is generally no history of rheumatism which might account for the heart murmur.

*On Examination:* The child is often stunted in growth, and he may be mentally backward. The fingers and toes are generally clubbed. The blood shows an excess of red cells and of hæmoglobin, the red cells numbering from 7 to 12 millions per c.mm., and the hæmoglobin may be as high as 150%. It is often difficult or impossible to diagnose the exact nature of the lesion, and more than one defect may be present. The typical findings in the more important congenital lesions will now be described.

### Congenital Pulmonary Stenosis

The patient may complain of dyspnœa, cough, hæmoptysis, headache or giddiness.

*On Examination.* There is cyanosis and clubbing of the fingers. The heart. Palpation. A systolic thrill may be felt in the second or third left spaces near the sternum. Percussion: The dulness is increased to the right. Auscultation. A harsh systolic murmur is

heard over the pulmonary base ; the pulmonary second sound is weak or absent.

### Patent Ductus Arteriosus

Frequently there are no symptoms, the patient is not cyanosed and the fingers are not clubbed. The heart. Inspection : Pulsation may be seen in the second and third left spaces, near the sternum, due to enlargement of the pulmonary artery. Palpation : A systolic thrill may be felt at the site of the pulsation, and a diastolic shock may be present. Percussion : A ribbon-shaped area of dulness (Gerhardt) may be found extending upwards from the pulmonary base to the left clavicle. Auscultation : A murmur may be heard, which is harsh or blowing, and begins just after the onset of the first sound. It fades away towards mid-diastole, and becomes loud again with each systole (water wheel murmur). The murmur is maximal at the second or third left space near the sternum, and it may be conducted towards the left clavicle, or heard at the back in the left interscapular region. It is due to blood flowing from the aorta to the pulmonary artery. The pulmonary second sound is accentuated. At the apex the second sound may be reduplicated. X-ray examination often shows a dilated conus arteriosus, and a pulsating ductus arteriosus may be detected on screening. Paradoxical embolus may occur, a clot passing from the left auricle or from the mitral or aortic valve to the lung. In some carefully selected cases ligation of the ductus has given excellent results in patients who have retarded physical development or evidence of cardiac embarrassment. One or two successful results have been published of combined operation and Sulphapyridine treatment in infected cases with a positive blood culture of streptococcus viridans.

### Patent Interventricular Septum

The heart. Auscultation : A loud or harsh murmur (*bruit de Roger*) may be heard, which starts early in systole and extends into diastole. It may be audible in the mid-sternal line, or along the left sternal border near the third costal cartilage, or lower down near the xiphisternum. It is not conducted to the neck or to the axilla. The pulmonary second sound is audible. The lesion may be associated with congenital heart block.

### Patent Foramen Ovale

Here a basal systolic murmur may be heard.

### Coarctation of the Aorta

Two types are described : 1. Adult. The constriction is at the site of the ductus arteriosus. 2. Infantile. There is narrowing of the isthmus between the left subclavian artery and the ductus arteriosus. The patient usually complains of no symptoms, but intermittent claudication may occur.

The heart. Inspection : The apex beat may be displaced a little outwards and downwards. Palpation : The apex beat is forcible. A

systolic thrill may be felt in the second left space, near the sternum. Percussion The right and left borders of the heart may be slightly "out." Auscultation Both sounds are heard at the apex and base. A systolic murmur is heard at the second left space, conducted downwards towards the apex. The blood pressure The pressure is raised in the arms, and low in the legs. The systolic brachial pressure may be 150 to 160 mm Hg in a child of 10 years, and often no pulsation can be felt in the femoral arteries. The arteries Enlarged and tortuous arteries may be seen or felt in the chest, especially in the interscapular region or in the neck and arms. X ray examination may reveal notching of the ribs due to the enlarged intercostal arteries (Roesler's sign). The ascending aorta dilates and the aortic knob is small. Death may result from congestive failure, rupture of the heart or aorta, cerebral hæmorrhage or bacterial endocarditis.

### Fallot's Tetralogy

This is a cause of cyanosis and clubbing of the fingers in adult life. A systolic thrill and murmur are usually present near the sternum in the second and third left spaces. The X ray findings are typical, the heart having a *cœur en sabot* appearance, the aorta is displaced to the right, the right ventricle is hypertrophied, and there is a concavity where the shadow of the pulmonary artery should be seen.

### Prognosis in Congenital Heart Disease

With certain lesions life is impossible as in *ectopia cordis*. With congenital pulmonary stenosis the patient usually dies before adult life, from some intercurrent disease, such as tuberculosis. With patent ductus arteriosus the patient may live well on into adult life. Infective endocarditis may be engrafted on a congenital valvular lesion and cause death. Sudden death may be due to cerebral hæmorrhage, embolus or rupture of the heart or aorta.

**Treatment.** There is no preventive treatment known. The patient usually requires to lead a sheltered life. As mentioned above, operation may be successful in certain cases of patent ductus arteriosus.

## ANEURYSM

**Definition.** An abnormal dilatation of an artery, resulting from changes in its walls.

**Varieties.** Aneurysms may be subdivided into 1. **True aneurysms.** The wall contains one or more of the arterial coats. They may be (a) **Fusiform or dilatation.** The aortic arch is especially liable to be thus affected. (b) **Saccular.** A localised swelling may form on large or small arteries. (c) **Dissecting.** A new channel is formed in the media through which the blood flows for a variable distance before regaining its normal path. (d) **Cirsoid.** A small artery and its branches are involved in a fusiform dilatation.

2. **Arterio-venous aneurysms.** These may be (a) **An aneurysmal varix.** There is direct communication between an artery and a vein.



(b) A varicose aneurysm. The artery and vein communicate through a sac.

3. False aneurysms. A swelling containing blood communicates with an artery, but the walls of the swelling are not formed by the arterial coats.

The fusiform, sacculated and dissecting aneurysms are of medical importance, the other varieties are chiefly of surgical interest. Small saccular aneurysms ("berry" aneurysms) may occur in the cerebral or retinal arteries, or elsewhere in the body. They are of clinical importance when occurring in the brain (see p. 298).

### Aortic Aneurysm

**Etiology.** Syphilis is the most important cause. The fusiform type, however, may be associated with arteriosclerosis and not with syphilis. A fusiform aneurysm may also develop in association with coarctation of the aorta. Cystic necrosis of the aorta is also a cause of dissecting aneurysm, which may subsequently rupture. Mycotic aneurysms occur in association with infective endocarditis and septicaemia, emboli being carried to the vasa vasorum of the arterial wall. Such mycotic aneurysms are usually multiple. *Predisposing causes:* 1. Age: Usually over 50. 2. Sex: Males especially. 3. Occupation: Involving strain.

**Pathology.** Syphilis causes a mesaortitis, the elastic and muscular fibres degenerate and the wall of the aorta yields to the pressure of its contents. The sac of the aneurysm is formed internally of layers of blood clot, outside which are the remains of the middle coat, the outer coat, fibrous tissue and perhaps surrounding structures to which it becomes adherent, such as the sternum, vertebrae or trachea. Calcareous plaques may be seen on its inner surface. In the adjacent parts of the aorta there are usually found the scars of syphilitic aortitis. In cystic necrosis of the aorta there is degeneration of muscular and elastic tissue in the media, with the formation of small cystic areas containing mucoid material. About 75% of aortic aneurysms are thoracic involving the arch, and about 10% are abdominal. The clinical findings of aortic aneurysms will be considered under separate headings, according to the portion of the aorta involved.

### Aneurysm of the Thoracic Aorta

Broadbent taught that an aneurysm of the ascending part of the arch is one of physical signs, whereas aneurysms of the transverse and descending parts of the aortic arch give rise chiefly to symptoms, due to pressure effects.

### Aneurysm of the Sinuses of Valsalva

**Clinical Findings.** The patient may be a comparatively young man, between 30 and 40 years of age. He may complain of faintness, precordial distress, headache or of pain resembling that of angina.

**On Examination:** Generally no signs of aneurysm are detected, but there is evidence of aortic reflu. In some instances, owing to pressure of the aneurysm on the inferior vena cava, there is oedema of

the legs, ascites, and enlargement of veins in the abdominal wall. Sudden death may occur from rupture into the pericardium. In other cases the rupture may be more gradual and of a dissecting character. The patient is seized with severe and prolonged pain, resembling that of a coronary obstruction. The radial pulses are not palpable, and no blood pressure can be recorded. The electrocardiogram is normal. After 4 or 5 days, sudden death ensues, the aneurysm finally rupturing into the pericardium.

### Fusiform Dilatation of the Aortic Arch

**Clinical Findings** The patient is usually an adult male past middle age. He may have no symptoms, or he may notice a little difficulty in breathing or in swallowing. In other cases the symptoms are those described on p. 246 as characteristic of aortic reflux.

**On Examination** **Inspection** The patient must be viewed in a good light both from the side and in front. Pulsation may be seen just above the manubrium sterni, or in the first or second right intercostal space close to the sternum, or the manubrium sterni itself may pulsate. **Palpation** The impulse may be felt at these sites. The cardiac apex is usually forcible, and it is often displaced a little downwards and outwards. A systolic thrill may be present over the aortic base, with a diastolic shock. **Percussion** An area of dulness may be detected over the manubrium sterni. **Auscultation** The aortic second sound may be clear and ringing, or an aortic diastolic murmur may be heard. There may also be an aortic systolic murmur. The arteries. These are usually thickened. Absence of the brachial and radial pulses on both sides has been recorded, probably owing to obstruction of the orifices of the innominate and left common carotid and subclavian arteries. The blood. The Wassermann reaction is not necessarily positive. **X-ray examination** Screening in the antero-posterior and oblique positions will reveal the dilatation of the aorta. It should be remembered that pulsation can often be felt in the suprasternal notch without any dilatation of the aorta being present.

### Saccular Aneurysm of the Ascending Arch

**Clinical Findings** The patient is usually an adult male, past middle age. He may give a history of syphilis years ago. The symptoms are very variable. Thus there may be no complaint of ill health, or the patient may notice pain in the region of the sternum on the right side of the chest, or in the back between the shoulders. He may complain of swelling of the face, neck, arms or hands, or of a feeling of engorgement of the face and neck, particularly on stooping. There may be dyspnoea on exertion or paroxysms of coughing. Slight stridor may have been noted when the patient is asleep. In some cases a throbbing is felt in the aneurysm, or hæmoptysis may be the first symptom.

**On Examination** **Inspection** The face and neck may be high coloured, with injected conjunctivæ, and one or both arms may be swollen. Enlarged veins may be seen on the front of the chest (see fig. 13). When the patient stoops down there is often considerable congestion of the

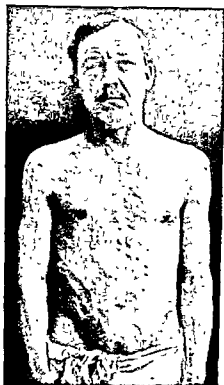


FIG. 13. DILATED VEINS ON TRUNK, RESULTING FROM OBSTRUCTION OF THE SUPERIOR VENA CAVA BY AN ANEURYSM OF THE ASCENDING PORTION OF THE ARCH OF THE AORTA.

A B

ASE OF ANEURYSM OF INNOMINATE ARTERY SHOWING A RIGHT HAND WITH CLUBBED FINGER  
AND B LEFT HAND FINGERS NORMAL



FIG 15 ANEURYSM OF INNOMINATE ARTERY  
The fingers of this patient are illustrated in Fig 14

face and neck. In rare cases, when there is interference with the blood supply to one arm, as in aneurysm of the innominate artery, unilateral clubbing of the fingers may be present (see Figs. 14 and 15). Slight pulsation may be detected in the second or third right space near the sternum, or there may be a definite pulsating swelling the size of a golf ball or larger. Palpation: Pulsation may be felt in the sites mentioned above, and with an aneurysm the pulsation may be expansile. A systolic thrill is rarely felt. The apex beat is usually forcible, and displaced a little downwards and outwards. Percussion: Dulness may extend outwards from the manubrium sterni to the right, in the first to third spaces, for 1 or 2 inches. Auscultation: The aortic second sound is accentuated, and a systolic murmur may be heard over the pulsating area. The pulse: The radial pulses may be unequal in force or in time, but this only occurs in about 4% of aortic aneurysms. A difference of blood pressure of over 20 mm. Hg. in the two arms is also of diagnostic significance. The lungs: Pressure on the right eparterial bronchus may result in slight dulness and weak air entry over the apex of the right lung. The trachea: Pressure here may cause dyspnoea, stridor (the leopard growl) or a harsh cough (gander cough). The right recurrent laryngeal nerve: This is rarely involved; pressure produces first abductor paralysis and later a complete paralysis. In the latter stage the cough is described as "bovine," lacking any explosive character. The blood: The Wassermann reaction is positive in a high percentage of cases, either before or after a provocative injection of neoarsphenamine.

### Saccular Aneurysm of the Transverse and Descending Arch

**Clinical Findings.** The patient is likely to complain of pressure symptoms. He may notice shortness of breath, either on slight exertion or when at rest, cough, expectoration, or difficulty in swallowing solid food. There may be pain under the sternum, in the back between the scapulae, round the chest or down the arms. There may be swelling of the face, the neck or the left arm. The dyspnoea may be relieved if the patient stoops forward, as this tends to increase the antero-posterior diameter of the chest.

**On Examination:** *Inspection:* Pulsation may be seen over the manubrium sterni or in the left interseapular region. *Palpation:* Pulsation or a thrill may be felt over the manubrium. *Percussion:* The heart may be enlarged downwards and to the left; dulness may be present over the manubrium. *Auscultation:* The aortic second sound may be accentuated. *Further signs of aneurysm:* A tracheal tug may be felt, if the cricoid cartilage is elevated with the forefingers, while the patient lifts up his head. The larynx may not move upwards with deglutition. The air entry may be weak over the upper or lower lobe of the left lung, and signs of bronchitis or of bronchiectasis may be found in the left lung. More rarely signs of distention of the left lung are noted, the note being hyperresonant and the breath sounds weak. Pressure on the left recurrent laryngeal nerve may cause laryngeal paralysis (see p. 121). The left pupil may be dilated from stimulation,

or constricted from paralysis of the sympathetic. There may be flushing or sweating limited to one side of the face, corresponding with the side of the aneurysm. Slowing of the heart may result from stimulation of the vagus. Pressure on the phrenic nerve may cause hiccough or paralysis of the left half of the diaphragm. Pain in the chest or left arm may be due to pressure on the intercostal nerves or the brachial plexus. The left pulse may be weaker than the right if the aneurysm is situated between the innominate and the left subclavian arteries, and the blood pressure lower in the left arm than in the right. Lowering of blood pressure in the left carotid artery may also cause dilatation of the left pupil due to relaxation of the vessels in the left iris. Pressure on the thoracic duct may result in chylothorax, and oesophageal pressure may cause dysphagia.

### Aneurysm of the Descending Thoracic Aorta

**Clinical Findings.** The patient may complain of a gnawing pain in the back in the thoracic region, radiating around the chest or spreading to the lumbar area. There may be pains in the legs or weakness, or later paralysis. Dysphagia, cough and expectoration are also sometimes present.

**On Examination.** Inspection. The back should be examined in a good light with the patient sitting up. It should be viewed from side to side when a pulsating area may be seen usually low down to the left of the thoracic vertebrae. Palpation. Pulsation or a thrill may be felt in the left interscapular region or just below this area. Percussion. The note may be impaired over this area. Auscultation. A systolic murmur may be heard in the left interscapular region. The arteries. The radial pulses are equal, but the femoral pulses may be weak or absent. The lungs. There may be signs of bronchitis or of bronchiectasis in the left lung or of a left pleural effusion. In more advanced cases erosion of the bodies of the vertebrae results in kyphosis or compression paraplegia with sensory and motor disturbances in the legs.

**Differential Diagnosis of Thoracic Aneurysms.** An aneurysm of the thoracic aorta may be very easy or very difficult to differentiate from a mediastinal tumour. An X-ray examination, despite the text books, is often inconclusive. A pulsating swelling may be seen, resembling an aneurysm which at autopsy is proved to be a new growth. In some cases an X-ray kymogram will help to differentiate, the pulsations of the aorta being distinguished from the transmitted pulsations of the tumour by the appearance of the edge of the shadow. The chief points in favour of the diagnosis of aneurysm are: 1. The age of the patient. This is usually over 40 years. 2. The sex. Males preponderate but an aneurysm may occur in a woman. 3. The condition of the patient. There is usually no cachexia, enlarged glands are not present, and the face often becomes congested on stooping. 4. The Wassermann reaction. This is positive in the majority of cases. 5. The course of the disease. Malignant growths are usually fatal within a year from diagnosis. 6. The X-ray appearances. These are at times conclusive. If the

aneurysm produces a definite swelling, visible in the chest wall, it must be distinguished from an abscess, a pulsating sarcoma or gunnma and an "empyema necessitatis." Expansile pulsation favours the diagnosis of aneurysm. Even a most experienced surgeon has been known to incise an aneurysm in mistake for an abscess.

**Course and Complications of Thoracic Aneurysms.** An aneurysm usually slowly increases in size; rarely it becomes obsolete by clotting, fibrosis or calcification. **Complications:** The aneurysm may rupture into the thorax, the pericardium, a bronchus, the œsophagus, or into the pleura, and rarely through the chest wall. An aneurysm may "weep," leaking for some time into a bronchus or elsewhere before rupture finally occurs. Other complications include the pressure effects detailed above.

**Prognosis.** A thoracic aneurysm is a very serious disease, and recovery is not to be expected. Death usually occurs between 4 and 15 years from the time of diagnosis.

**Treatment. Prophylactic.** Syphilis should be treated adequately and early.

**Curative.** The patient should be kept in bed for at least 3 months after the diagnosis is made. This will aid clotting by diminishing the circulation rate. If there is venous engorgement, venesection of 10 to 20 oz. from the arm often affords much relief, and can be repeated as required. The diet should be moderate, and if the blood pressure is high, meat and stimulants are best avoided. A regular daily action of the bowels should be secured with the help of salts, senna or cascara sagrada. If the Wassermann reaction is positive, a course of potassium iodide and mercury should be given. The iodide is increased from gr. 5 to gr. 30 t.d.s., and given for 3 months. Mercury can be administered either as liq. hydrarg. perchlor. m. 20 to 60 t.d.s. in the iodide mixture; or as an inunction, gr. 60 of blue ointment (ung. hydrarg. B.P.) rubbed into the skin of the axillæ and groins for 4 nights in different sites, avoiding areas where hair is growing, it is then omitted for the next 3 nights; or by 12 weekly injections into the buttock of a mercurial cream, containing gr. 1 of mercury; or as a pill composed of hydrarg. c. cret., and pulv. ipecac. et opii aa gr. 1, one pill twice a day. Subsequently a course of *Quinostab* and *necoursphenamine* is given, providing there are no anginal symptoms (see p. 248). The pain is generally relieved by the iodides, but if very severe, *Nepenthe* m. 10 to 15 may be required. When the patient is allowed up, all violent exercise or strain must be avoided.

### Aneurysm of the Abdominal Aorta

Saccular, fusiform or dissecting aneurysms may develop. A saccular aneurysm near the origin of the cœliac axis artery is perhaps the most common variety.

**Clinical Findings.** The patient complains of abdominal or lumbar pain. It is a continuous type of pain, and may be intensified by lying down.

**On Examination:** Inspection: With a large aneurysm a pulsating

swelling may be seen in the epigastrium, or at times in the back near the lumbar vertebræ. **Palpation.** A pulsating swelling is felt in the abdomen. This is definitely expansile. The femoral pulses may be obliterated. In some cases an aneurysm may form on a renal artery, with the appearance of a large pulsating swelling in the loin.

**Differential Diagnosis.** A pulsating abdominal aorta is frequently noted in thin patients, especially in women. It does not usually cause pain, although its throbbing may produce discomfort. The swelling is not expansile, the femoral pulses are normal and there are no pressure symptoms, such as may result with an abdominal aneurysm from erosion of the vertebræ. An abdominal tumour may receive transmitted pulsation from the aorta. If the patient is examined in the knee-elbow position, the pulsation usually disappears as the tumour drops forward.

**Treatment.** This is as for thoracic aneurysm.

### Dilatation of the Pulmonary Artery

**Etiology.** Dilatation of the pulmonary artery is a rare disease usually associated with a congenital lesion of the heart, such as a patent ductus arteriosus with a superadded infective endocarditis of the pulmonary artery. It may also be met with in association with pulmonary arteriosclerosis (see p. 264).

**Clinical Findings.** The patient is usually a young adult. When infective endocarditis is present, the symptoms are chiefly due to this lesion, such as malaise and fever, and perhaps recurrent hæmoptysis.

**On Examination.** There is slight cyanosis and some clubbing of the fingers. The heart. **Inspection.** Pulsation is seen in the second and third left spaces near the sternum. The apex beat may be visible, displaced a little outwards. **Palpation.** A systolic thrill is felt over the pulsating area at the pulmonary base. **Percussion.** The cardiac dulness is enlarged to the right and to the left, and it extends upwards and outwards over the pulsating area. **Auscultation.** A systolic murmur is heard over the pulmonary base. The pulmonary second sound is accentuated. A variable diastolic murmur may be present along the left sternal border. **X ray examination.** The heart shadow is increased transversely. The aortic shadow is normal. An enlarged pulsating shadow is seen in the region of the conus arteriosus.

**Differential Diagnosis.** The enlarged pulmonary artery is differentiated from a dilated left auricle, as the latter enlarges backwards and to the right.

**Treatment.** This is merely symptomatic for cough and dyspnoea. There is no curative treatment.

### Acute Polyarteritis Nodosa

#### (Periarteritis Nodosa)

**Definition.** A condition characterised by small localised aneurysms on the medium sized arteries.



**Etiology.** Acute polyarteritis nodosa is probably caused by bacterial infection.

**Pathology.** There are perivascular inflammatory changes. The middle arterial coat is destroyed at localised areas, with resultant aneurysmal dilatation. These small aneurysms may be found in the heart, kidneys, mesentery, liver, stomach, intestines, spleen, diaphragm, brain, lungs, muscles, and under the skin.

**Clinical Findings.** The patient is usually a male between the ages of 30 and 40, but he may be a young child. The onset of symptoms is generally sudden, with fever, weakness, muscular or joint pains, vomiting and diarrhoea. In other cases there is a sudden onset with coma and convulsions, or the illness may be characterised by severe abdominal or cardiac pain, hæmoptysis, or hæmaturia. Bronchial asthma may be a prominent symptom. *Urticaria and purpura may also occur.*

**On Examination:** Small nodules may sometimes be felt on the subcutaneous arteries of the chest or abdomen. The urine may contain blood or albumin. The blood pressure is often raised. The temperature is usually raised, and the blood may show a leucocytosis of about 30,000 per c.mm. In some cases there is eosinophilia.

**Differential Diagnosis.** The nodules may suggest somatic tæniasis. The fever and abdominal symptoms may resemble those occurring in enteric fever or in miliary tuberculosis. The nervous symptoms are liable to be mistaken for meningitis or a cerebral hæmorrhage. Acute renal pain may suggest a renal calculus or perinephric abscess. The hæmaturia suggests acute nephritis. The dyspnoea with eosinophilia may be mistaken for asthma. It is rare for a correct diagnosis to be made before autopsy, unless the nodules are felt attached to the subcutaneous arteries.

**Course and Complications.** The disease usually pursues a progressive course lasting 3 to 4 months, but cerebral hæmorrhage may cause sudden death.

**Prognosis.** The disease is generally fatal.

**Treatment.** This is symptomatic, a blood transfusion is not of any permanent value. A course of intravenous injections of neoarsphenamine should be tried (see p. 570).

### Arteriosclerosis

**Definition.** Local (focal) or diffuse hardening of arteries, due to inflammatory or degenerative changes in their coats.

**Etiology.** Arteriosclerosis is associated with many diseases. The exciting causes include acute infections such as scarlet fever, enteric fever, rheumatic fever, the presence of septic foci with chronic bacterial toxæmia, or infection with syphilis or tuberculosis. *Predisposing causes:* Old age, high blood pressure, chronic nephritis, overwork, overeating, gout, diabetes mellitus and a hereditary diathesis. The following varieties are described:

1. *Atheroma* (nodular arteriosclerosis). This is usually associated with a raised blood pressure, and hypercholesterolaemia may be present.

It can be induced experimentally in rabbits by feeding with a diet rich in cholesterol

2 *Arterio capillary Fibrosis* (Gull and Sutton), or diffuse hyperplastic sclerosis This may be due to supertension (Jores), to inflammation (Virchow) or to involution (Thoma) It is generally considered to be inflammatory, an arteritis induced by bacterial toxins

3 *Senile Arteriosclerosis* This results perhaps from chronic toxæmia and old age

4 *Monckeberg's Sclerosis* This may occur in younger people, associated with carcinoma, tuberculosis, heart disease, chronic infections and diabetes mellitus

5 *Mesaortitis* This is due to syphilis

6 *Endarteritis Obliterans* This may be due to syphilis or to tuberculosis

7 *Periarteritis* This may occur in the arterioles of the brain or cord in association with syphilis, polioencephalitis, encephalitis lethargica or tuberculosis

**Pathology Atheroma** A localised thickening of the intima results in the formation of yellowish white plaques The aorta and large arteries are chiefly affected, also the coronary and cerebral vessels, and those supplying the lower limbs rather than the arteries of the arms Fatty degeneration may lead to an atheromatous ulcer, and calcification may ensue Microscopically there is proliferation and fatty degeneration of the endothelial cells and proliferation of the connective tissue cells

*Arterio-capillary Fibrosis* The small and middle-sized arteries, arterioles and capillaries are chiefly affected, and there is usually some phlebosclerosis Hyaline degeneration occurs in the intima of the arterioles, especially in the kidneys, spleen, brain, pancreas, liver and suprarenals The parent arteries show intimal thickening from proliferation of the subintima In the capillaries the endothelium swells and degenerates Fibroid myocarditis and chronic nephritis are often present in adults, and renal disease in children (see also p. 400)

*Senile Arteriosclerosis* The medium and smaller sized arteries are affected Fatty degeneration and calcification of the media occurs, with the formation of pipe stem vessels The intima is thickened

*Monckeberg's Sclerosis* Here fatty degeneration and calcification of the media occur, but often there is no intimal thickening

*Mesaortitis* Pearly patches are found in the aorta, especially near the heart They are leathery to the touch, with a pitted surface and crenated outline They do not usually undergo calcification Depressed linear scars may be seen in some instances Microscopically there is round celled infiltration of the vasa vasorum, with degeneration of the medial musculature, and spirochaetes may be demonstrated.

*Endarteritis Obliterans* The smaller arteries and arterioles are affected, with thickening of the intima. This occurs especially in the brain and in the vasa of the aorta

*Periarteritis.* The adventitia hypertrophies and may degenerate around the arterioles of the brain and cord, spirochaetes may be found in them in some cases.

**Clinical Findings.** The patient is a child or an adult; usually, however, a male past middle age. The onset of the disease is insidious and nothing abnormal may be noticed, beyond a slight pallor in some cases, until the arterial changes produce symptoms definitely located to some portion of the body. In other cases, when there is a diffuse thickening taking place in the various arteries of the body, the patient may complain that he is becoming prematurely old and unfit for the activities to which he has been accustomed. The effects produced by arteriosclerosis vary with the type of pathological change present, but clinically they are best considered regionally. The blood pressure is usually raised and the heart hypertrophied, but in senile arteriosclerosis this is often not the case, and it is not necessarily so in atheroma. The condition of the arteries may be judged clinically by palpating those which are superficial, such as the radial, brachial, facial, temporal, femoral and dorsalis pedis. The radial artery may be firm like a whipcord in diffuse hyperplastic sclerosis, or calcified nodules may be felt in atheroma, or it may be hard like a pipe-stem, in the senile or Mönckeberg's types. The radial and brachial arteries are sometimes tortuous, and this may occur without calcification. Tortuosity of the temporal arteries does not necessarily imply arteriosclerosis. Ophthalmoscopic examination may show arteriosclerotic changes in the retinal vessels.

**Cerebral Symptoms.** There may be mental deterioration, lack of concentration and insomnia; or attacks of giddiness, temporary loss of consciousness, paresis, aphasia or epileptiform convulsions may occur. Arteriosclerotic Parkinsonism is also described, characterised by rigidity, affecting especially the trunk and lower limbs, a mask-like expression, and bradykinesia. Transitory blindness due to spasm of the vessels may ensue. Cerebral thrombosis or hæmorrhage are more serious complications.

**Cardiac Symptoms.** Arteriosclerosis of the aorta or coronary arteries may lead to aneurysm of the aorta, aortic regurgitation, myocardial degeneration, cardiac hypertrophy, angina pectoris, coronary thrombosis, or aneurysm of the heart followed by rupture.

**Abdominal Symptoms.** Attacks of abdominal pain, constipation or mesenteric thrombosis may ensue.

**Renal and Vesical Symptoms.** Arteriosclerotic kidneys may result in chronic nephritis, usually without œdema (see p. 460). Hæmaturia may result from arteriosclerosis of vessels in the bladder.

**Peripheral Symptoms.** Involvement of the arterial supply of the extremities, especially the legs, results in intermittent claudication. The patient finds he cannot walk more than a few yards without severe pain in the legs, which ceases as soon as he rests. In more advanced cases there may be pain and cramps apart from exercise, and gangrene may slowly set in. In the diffuse hyperplastic sclerosis the clinical picture is that of a systolic blood pressure of 180 mm. Hg. or more, with secondary cardiac hypertrophy (hypertensive heart disease). This is known clinically as hyperpiesia or essential supertension (see p. 265).

**Differential Diagnosis.** Arteriosclerosis is usually easily diagnosed by palpating the peripheral vessels. In the senile form, an X-ray film

may show the calcification. Intermittent claudication due to arterio sclerosis must be differentiated from thrombo angutis obliterans (see p 269). The characteristic features of the latter disease are that it occurs in younger men, usually Hebrews, and migrating phlebitis is also present in the legs.

**Course and Complications.** The arterial changes are progressive, and such complications as hæmorrhage, gangrene or muscular degeneration are liable to occur from interference with the blood supply to the tissues. Bronchitis may develop.

**Prognosis.** The outlook is more unfavourable when calcification occurs in the arteries of a young man. Myocardial degeneration, aneurysm and renal inefficiency all increase the severity of the condition.

**Treatment.** This is in the main that of high blood pressure (see p 267) i.e., elimination of septic foci, an efficient daily evacuation of the bowels, attention to any digestive disturbance, moderation in diet, especially in proteins, avoidance of alcohol and smoking, restriction of exercise, freedom from mental fatigue and worry, and the observance of regular hours.

In syphilitic cases a course of anti syphilitic treatment should be given with iodides, mercury and Quinostab. Neoarsphenamine may be used subsequently, providing there are no angular symptoms (see p 248). For intermittent claudication relief has been obtained in some cases by passive vascular exercises (see p 271) or by lumbar sympathectomy. Apart from this, rest, massage and diathermy afford relief at times. Amputation is usually required if there is gangrene. Treatment with muscle extracts or with nucleosides from body tissues is of doubtful value.

### **Pulmonary Arteriosclerosis**

(*Pulmonary Atheroma* *Ayerza's Disease* *Cardiacos Negros*)

**Definition.** Sclerosis of the pulmonary artery, associated with erythrocytosis and severe cyanosis.

**Etiology.** There are two main groups of cases. 1. Due to primary changes in the pulmonary artery and its branches. 2. The changes in the pulmonary artery are secondary to changes in the lungs and heart, such as chronic bronchitis, emphysema and mitral stenosis. The primary form is usually due to a syphilitic infection of the pulmonary artery, but more rarely there is a non inflammatory sclerosis occurring in younger people the cause of which is unknown. Ayerza's cases were secondary to long standing disease of the lungs.

**Pathology.** The heart is enlarged, the right ventricle being dilated and hypertrophied. The intrapulmonary branches of the pulmonary artery show obliterative arteritis in the syphilitic cases, the pulmonary artery is dilated and atheroma is seen in the main right and left branches. The lungs usually show fibrosis, bronchitis and emphysema. There is passive hyperæmia of the liver.

**Clinical Findings.** The onset is insidious, the patient complaining of gradually increasing dyspnoea, followed by cough, expectoration and

at times recurrent hæmoptysis. He may also notice palpitations, giddiness, and somnolence.

*On Examination:* The most striking feature is the deep cyanosis of the face and neck. With primary arteritis of the branches of the pulmonary artery there are often no other definite physical signs. If there are changes in the lungs the fingers are usually clubbed. In a more advanced case the heart is enlarged both to the right and the left. A radiogram shows marked prominence of the pulmonary artery (bulging of the left middle arc), the heart being rotated to the left the base of the pulmonary artery is well shown. Pulsating shadows of the pulmonary artery may be seen at the roots of the lungs, and the shadows of the intrapulmonary branches of the pulmonary artery may also be evident. The electrocardiogram shows right axis deviation. The blood: The red cell count varies between 5 and 9 millions. The Wassermann reaction may be positive. In the later stages there is ascites, œdema of the legs and enlargement of the liver.

*Differential Diagnosis.* Erythrocytosis (see p. 514) may occur in association with many conditions, such as congenital heart disease, chronic bronchitis and emphysema, or polycythæmia rubra. The diagnosis is usually established by the X-ray appearances.

*Course and Complications.* The course is progressive, complications include right-sided heart failure or intercurrent infections, such as bronchopneumonia.

*Prognosis.* This is grave, death usually occurs within 5 years from the date of diagnosis.

*Treatment.* Anti-syphilitic treatment (see p. 248) should be given if the Wassermann reaction is positive. Apart from this, venesection is of value in relieving the right-sided cardiac embarrassment, other treatment being symptomatic.

### Phlebosclerosis

Hardening of the veins may occur in association with varicose veins, in thrombo-angiitis obliterans, in arterio-capillary fibrosis, and in diabetes mellitus. The pulmonary veins may be affected in mitral stenosis.

### Thrombosis of the Axillary Vein

This may occur in healthy young adults as the result of a sudden strain made with the arm and hand; rarely it develops spontaneously. It may be due to pressure of the costocoracoid ligament and subclavius muscle when the arm is abducted. The affected arm swells and is cyanosed, and dilated superficial veins appear on the arm, in the axilla and over the anterior part of the chest. The arm should be rested, elevated, and local diathermy treatment applied.

### High Blood Pressure

#### (*Hyperpietis. Supertension*)

*Definition.* Increased blood pressure from any cause.

*Physiology and Pathology.* The maintenance of blood pressure

depends upon four factors, modifications of which may cause it to rise. These factors are the heart, the arteries, the peripheral resistance and the blood.

*The cardiac factor*—Supertension may be associated with cardiac hypertrophy as in aortic disease, mitral regurgitation, adherent pericardium or Graves' disease.

*Arterial factors*—Arterial obstruction, arteriosclerosis and arterial spasm associated with their various causes such as essential hypertension, coarctation of the aorta, acute and chronic nephritis, hydronephrosis, congenital cystic disease of the kidneys, myxoedema, Cushing's syndrome, angina pectoris, tabetic crises, migraine, and possibly toxæmia, etc.

*Peripheral factors*—The tone of the arterioles and the capillary peripheral resistance may be increased by irritability of the vaso-motor system.

*Hæmic factors*—An increased blood volume may be associated with overeating, overdrinking, and possibly with salt retention in renal disease. Increased viscosity of the blood occurs in the polycythæmia hypertonica of Gaisböck (see p. 514).

Supertension may be temporary or permanent. Transitory supertension may result from emotion, pain, exercise, angina pectoris, a tabetic crisis or acute nephritis. Emotion raises the systolic and to a lesser degree the diastolic pressure. The blood pressure is not altered by height and weight provided they are proportional. Permanent supertension may be met with in the young, middle aged or elderly. It is often discovered on routine examination without the patient having any symptoms. The normal pressure varies at different ages but in health there is very slight change between the ages of 18 and 40, the average reading for this age period being 122 mm Hg systolic and 76 mm Hg diastolic. A low pulse pressure with a normal systolic reading is probably pathological. A diastolic pressure which is higher than 120 mm Hg is of serious significance and in a young adult a persisting diastolic pressure over 90 mm Hg is usually abnormal.

### Essential Hypertension

(*Hypertæsis* (Allbutt) *Hypertensi e Cardio vascular Disease* (Janeway))

**Definition.** A condition of hypertension of unknown origin, usually not associated with demonstrable renal insufficiency.

**Etiology.** The cause is unknown. Hypertension can be produced in dogs by partial constriction of the renal arteries. A pressor substance, renin, is thought to be produced in excessive amounts in the ischæmic kidneys. This work suggests that essential hypertension is due to a toxic substance circulating in the blood, and produced in the kidneys whose blood supply is deficient. The high blood pressure does not result in any increase of general circulatory efficiency.

**Predisposing causes.** 1 Age 30 to 50 years. 2 Sex Females predominate slightly. 3 Heredity. There is a definite familial incidence.

**Pathology.** Post mortem examination usually reveals cardiac hypertrophy, the left ventricle being affected. Various arterioles in the body show arteriolosclerotic changes especially in the kidneys, spleen, pancreas, liver and brain. The kidneys may show granular changes with an adherent capsule.

**Clinical Findings.** Early signs may at times be found in children. Thus in a child the pressure may be a little above normal at rest. After exercise the pressure may rise considerably, taking 20 minutes or longer, instead of the normal 5 minutes to return to its resting value. Adult patients usually seek advice for such symptoms as dyspnoea on exertion, palpitations, a sense of præcordial oppression, inability to sleep on the left side, headaches, giddiness, tinnitus aurium, lack of concentration, irritability, anginal pains, epistaxis, numbness or tingling in the legs, cramp or coldness of the legs, frequency of micturition at night, transient blindness, a stroke or rarely for hæmoptysis.

**On Examination:** The clinical findings are those described under hypertensive heart disease (see p. 223). The blood pressure should be determined with the patient lying down, the systolic and diastolic readings being taken both by the palpatory and the auscultatory methods, and repeated 2 or 3 times or after a few days' interval, until constant readings are obtained, in order to allow for the abatement of nervous excitement and arterial spasm. Readings should be taken on both arms, as there is frequently a difference of 5 to 10 mm. on the two sides. A more marked discrepancy suggests the presence of an organic cause, such as a cervical rib, aneurysm or mediastinal tumour.

**Course and Complications.** Cases of hyperpiesia often show irregular variations of blood pressure from time to time; crises may occur in which the blood pressure suddenly falls and the patient is collapsed or even suffers from convulsions and unconsciousness. Hypertensive cerebral attacks are also liable to occur, these were formerly considered as uræmic (see p. 465). They are due to a rapid rise in the blood pressure. In one form, which is more prone to develop in patients under the age of 40, there is cerebral œdema, with severe headache, vomiting, drowsiness and possibly coma. The retinal arterioles are constricted early in the attack. The diastolic pressure may rise to 160 mm. Hg. The blood nitrogen figures are normal, unless the kidney function also has failed. The second variety is more common in individuals over the age of 40, and is probably due to angiospasm. The patient suddenly becomes unconscious in an epileptiform attack. This may be followed by coma, or there may be transient aphasia, monoplegia or hemiplegia. When associated with renal disease the course is usually progressive (malignant essential hypertension). In some cases there is a unilateral renal lesion, such as tuberculosis or a hypernephroma, and nephrectomy is followed by the restoration of a normal blood pressure. Complications are those of the associated cardio-vascular and renal lesions, such as cerebral hæmorrhage or uræmia.

**Prognosis.** This depends upon the height of the pressure and the state of the heart and arteries. A high diastolic reading is always of grave significance. The average duration may be stated to be 10 years from the onset of symptoms. The prognosis is more favourable in men than in women. The younger the disease shows itself, the more serious is the outlook.

**Treatment.** In the permanent cases of supertension a definite regime should be laid down. There must be adequate rest and

freedom from mental and physical strain. The diet. Beef and mutton should be avoided, poultry, fish, vegetables, fruit and eggs are allowed. The patient is best without alcohol, and smoking should be very moderate. A sufficient amount of fluid should be taken to allow for the excretion of about 3 pints of urine daily. A glass of hot water may be taken before breakfast and on retiring to bed. The bowels should be kept open daily, with a small dose of Epsom salts, such as gr 30 to 60 every morning, and once a week calomel, gr 1 to 3 or pil hydrarg, gr 4, should be taken at night. Any septic focus in the mouth or elsewhere should be treated. The Wassermann reaction should be determined, and if positive a course of treatment given with iodides and mercury, and subsequently with neoarsphenamine (see p 248). Various drugs may be prescribed, such as sodium iodide in doses of gr 5 to 10 tds for periods of 6 weeks, with a month's intermission. Other hypotensive drugs may be used, such as tab glyceryl trinitrat gr 1/120 tds tab sod nitrit co (B P C) 1 tds, or an organic preparation of iodine such as Lipoiodine Ciba tablets (gr 5), one one day and two the next day alternately. In some cases good results are obtained with bism subnitrat (B P C) gr 10, in a capsule tds. A course of Spä treatment is often beneficial, as the patient should then be compelled to carry out strictly his proper regime. In those cases of hyperpiesia associated with severe headaches and vomiting a course of autohæmotherapy is of value. Five to 10 mls of blood are removed from a vein in the patient's arm and immediately injected into the gluteal muscles. This is repeated once a week for 6 injections. The treatment for hypertensive cerebral attacks by venesection, lumbar puncture and hypertonic saline solutions, is described on p 166 (see Acute Uremia). In all cases of supertension the use of a simple enema at night followed by a colonic injection of 8 oz of 25% mag sulph solution the next morning is advisable. This often lowers the pressure 10 to 20 mm Hg and can be repeated at intervals of 5 to 7 days, according to the permanency of the result obtained. Bilateral splanchnicectomy may be beneficial in a small number of cases. The main indications are severe hypertension, a blood pressure of 200/120 mm Hg or more, which does not improve with rest, and an absence of renal or myocardial disease or of generalised arteriosclerosis. Further, the blood pressure should not be fixed  $\pm$ , it should approximate to normal during sleep or under light Pentothal Sodium anaesthesia.

### Low Blood Pressure

#### (Hypopiesis Subtension)

**Definition.** Diminished blood pressure from any cause.

**Etiology.** Low blood pressure may be due to disease of the suprarenals, as in Addison's disease and possibly in diphtheria and other acute infections. It may result from capillary dilatation due to liberation of a histamine body in anaphylaxis or intestinal obstruction. Other causes include severe loss of blood, surgical shock, chronic cachexia or sepsis, myocardial degeneration, coronary obstruction and diabetic coma.



**Pathology.** The low blood tension is usually due to capillary dilatation, and in some cases the blood is pooled in the splanchnic area.

**Clinical Findings.** A low blood pressure may be an acute and temporary state, which shows itself by fainting and loss of consciousness. In other cases it is persistent, the patient complaining of lassitude, insomnia, headache and giddiness. In some cases there are no symptoms, as 3% of over 2,000 fit air pilots in England were found to be hypotensive.

**On Examination :** The blood pressure in an adult is considered to be low if the systolic reading is below 110 mm. Hg.

**Treatment.** The blood pressure can be raised very temporarily by a subcutaneous injection of liq. adrenal. hydrochlor., m. 8 to 16. Ephedrine sulphate tablets, gr.  $\frac{1}{2}$ , may be given by mouth, t.d.s. The treatment of Addison's disease is considered on p. 666.

The patient should have an adequate amount of rest, his dietary should be mixed and contain fresh foods, and he should drink at least 3 pints of fluid daily. For anæmia iron or liver extract should be given according to the type of anæmia present. If the low blood pressure is associated with myocardial weakness, a course of digitalis should be given, such as m. 5 to 10 of the tincture t.d.s. for 3 or 4 weeks. Low blood pressure due to hæmorrhage or shock is treated by warming the patient with an electric cradle and giving intravenous injections of normal saline, whole blood or plasma.

### Thrombo-angiitis Obliterans

(*Thrombotic phlebo-arteritis. Buerger's Disease. Von Winiwarter's Disease. Die Hebraische Krankheit*)

**Definition.** A disease characterised by thrombosis of portions of arteries and veins in the extremities, with an inflammatory reaction around the affected vessels.

**Etiology.** The cause is unknown. It is thought to be due to absorption of bacterial toxins, perhaps from the prostate or the intestines. It is not due to syphilis. *Predisposing causes :* 1. Age : 25 to 50. 2. Sex : Chiefly males. 3. Heavy cigarette smoking. 4. Race : Chiefly Jews.

**Pathology.** Thrombosis occurs in the larger arteries of the legs and in the superficial veins. There is some organisation of the clot, and an inflammatory reaction around the vessels with fibrous tissue formation, which may also involve the nerves.

**Clinical Findings.** The patient is usually a male between the ages of 25 and 50 years. The onset often occurs in cold weather, with numbness, coldness, or pain in the foot or leg, induced by walking and relieved by rest. Later the pain becomes more severe and occurs apart from exertion (rest pain). After a year or so the other leg is affected and later it may spread to the fingers and hands. Sleep may become impossible owing to pain, which is intensified when the leg is elevated. Painful spots may also be noticed which are due to superficial venous thrombosis.

*On Examination* • In a developed case the leg is red and shiny when dependent, and pale on elevation or on lying down, no pulsation can be felt in the dorsalis pedis or posterior tibial arteries. The foot or leg feels cold and the skin temperature is lower than that of the unaffected leg. With an oscillogram accurate readings can be obtained of the difference in the blood pressure in the two legs, the cuff can be placed above the knee and ankle. Thrombosed superficial veins may be seen and felt, their site varying from time to time. There may be small blisters containing bloodstained fluid on the toes, ulcers often form under the big toe nail, and œdema of the foot, or actual gangrene of the toes may ensue. There is no muscular wasting and no sensory changes are present. The determination of the skin temperature and the response to heat or to a spinal anæsthetic is of great importance in deciding the amount of vasoconstriction which can be overcome by sympathectomy. It is an index of the amount of muscular tissue as compared with fibrous tissue in the arteries. Alternatively it may indicate the degree to which the circulation is increased owing to vasodilation of the healthy vessels. The blanket method is a convenient way of performing the test. The patient lies naked except for a loin cloth, in a room with a constant temperature of 78° F for an hour. The skin temperature is then taken, with a special mercury thermometer or a thermocouple connected with a delicate galvanometer, at the following points: Mouth, axilla, elbow, wrist, interdigital spaces, tips of digits, groin, knee, ankle, interdigital spaces, tips of digits (on both sides of the body). The patient is then wrapped, except for his face, in a warmed light rubber sheet, and covered by three blankets. After one hour the skin temperatures are again taken at the same points and the results are recorded on a graph. Normally, or if there is little vasospasm, the second graph is almost a straight line, at or slightly below the mouth temperature level. When a limb has arterial obstruction the skin temperature is some degrees lower than that of a normal limb, and, on raising the temperature around the body the rise in skin temperature is proportional to the capacity of the superficial vessels to dilate (see Fig. 16 facing p. 272).

*Differential Diagnosis* The disease must be differentiated from Raynaud's disease, erythromelalgia, arteriosclerotic endarteritis obliterans, thrombophlebitis migrans, popliteal aneurysm, neuritis, flat foot and rheumatism. In Raynaud's disease there is no dependent rubor, no venous thrombosis, and the arterial pulse is present. In erythromelalgia there is no phlebitis, no claudication, no gangrene, the arteries pulsate normally, and the limb does not blanch markedly on elevation. In arteriosclerotic endarteritis calcification of the arteries can be felt, or seen with the X rays, there is no migrating phlebitis, and the age incidence is usually later. In thrombophlebitis migrans there is no claudication and no arterial obliteration. It is possible, however, that some cases are early stages of thromboangiitis obliterans. Many cases are diagnosed in the initial stages as flat foot or rheumatism, and so much valuable time is lost.

*Course and Complications* The course is usually prolonged for 10 years or longer, there is a tendency to spread from limb to limb,

but pulsation may return in the affected artery. Acute fulmination may occur with œdema and gangrene of the limb.

**Prognosis.** This is unfavourable; death is usually due to an intercurrent disease.

**Treatment.** At the onset the patient should be kept in bed and smoking forbidden. Any septic focus should be treated. Local treatment consists in passive movements to the leg. It is raised to produce blanching for 3 minutes, and then hung over the edge of the bed to effect rubefaction for 5 minutes, and finally it is kept horizontal for another 5 minutes. This exercise is repeated several times daily. Passive vascular exercises can be given by the Pavax apparatus, the limb being placed in a glass case, inside which the pressure is rhythmically changed from  $-80$  mm. Hg. to  $+20$  mm. Hg., two, three or four times a minute, for periods of 20 minutes daily. Intermittent venous occlusion can also be used. The cuff of a sphygmomanometer is fixed on the thigh and the pressure is varied, by an automatic apparatus, from 50 mm. Hg. for 1 minute to zero for 2 minutes. The treatment is given for half an hour daily. Intravenous hypertonic saline injections are successful in some cases, 300 mls of 3% NaCl solution being injected intravenously every other day. As an alternative to this the patient may drink daily 3 to 4 quarts of water containing 10 G. of table salt to 1 quart. Non-specific protein therapy is also employed, injecting intravenously 1 million organisms of typhoid and paratyphoid A and B. If this produces fever or malaise, the second dose should be reduced to 100,000 organisms. Muscle extracts have proved beneficial in the treatment of some cases, a preparation such as Carnacton being used. 1 to 2 mls are injected intramuscularly three times a day for several months. The beneficial results of such treatment can be seen by the skin temperature curve in Fig. 16. Favourable results have also been obtained by the use of sex hormones. Testoviron, mg. 25 in 1 mil., is injected intramuscularly daily for 2 to 3 weeks in the case of a male, and Progynon B. oleosum, mg. 1 (10,000 units) for a woman. The injections are given daily for a week and every other day during the second week. Surgical treatment consists in lumbar sympathectomy with bilateral removal of the 2nd to 4th L. ganglia. The operation will only remove spasm, and should therefore only be employed if the skin temperature tests indicate that the vessels are still capable of dilatation. In cases of gangrene or pain unrelieved by other methods, amputation alone can afford relief.

### Thrombo-phlebitis Migrans

**Definition.** Phlebitis of the superficial veins, recurring in different sites and often accompanied or followed by venous thrombosis in internal organs.

**Clinical Findings.** Recurrent attacks of phlebitis occur usually in the legs. The patient may have symptoms suggesting thrombosis in the lungs, the abdomen, the heart or the brain. Thus he may be attacked with acute pain in the chest, and cough up some rusty sputum. or he may have severe abdominal pain, or cardiac pain or symptoms

a cerebral thrombosis. The temperature is usually elevated with each recurrence of thrombosis and the blood shows a leucocytosis.

**Prognosis** This is usually favourable, even when thrombosis occurs in internal organs, although there is a tendency to recurrence.

**Treatment** The patient must be kept in bed. Plenty of fluids should be taken by mouth, and a mixture containing Sod citrat gr 30, syr aurant m 20 aqua ad ii oz 1 is given every 6 hours. Subsequently the teeth should be X rayed and any septic focus eradicated. A bacteriological examination of the urine and faeces should also be made and if pathological organisms are found an autogenous vaccine should be administered.

### Erythromelalgia

(Weir Mitchell's Disease)

**Definition** A disease characterised by redness, heat and pain of the extremities, especially the feet, which are worse when they are dependent.

**Etiology** The cause is unknown. It may be associated with exposure to cold and wet, or with much walking.

**Pathology** The vascular changes may be due to a disorder of the sympathetic nervous system.

**Clinical Findings** The patient is usually an adult woman between the ages of 30 and 50. She first notices pain in the soles of the feet after standing or walking. The pain is of a burning character and later may be very severe, occurring also in bed (rest pain) when the feet are hot. It is usually worse in the summer but there is no claudication.

**On Examination** One or both feet are red, and the lower half of the leg may also be affected. The foot feels hot. Pain is experienced when the surface temperature reaches 34° C. There is no oedema and no gangrene. The foot does not become much more red when dependent, nor does it blanch considerably on elevation. The arterial pulsation and sensation are normal.

**Differential Diagnosis** Erythromelalgia must be differentiated from Raynaud's disease and painful rubor associated with thromboangitis obliterans and arteriosclerosis affecting the extremities. The differential diagnosis is considered on p. 270.

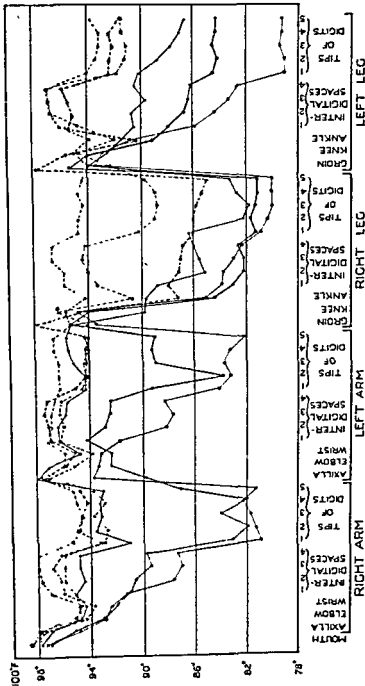
**Course and Complications** The disease is usually progressive, but complications are rare. Both hands and feet may eventually be affected.

**Prognosis** The chance of spontaneous cure is remote and the disease usually persists for many years.

**Treatment** Early and prolonged rest is of the greatest importance. Pain may be relieved by cold applications, but if it is intense lumbar cord ganglionectomy is the most hopeful method of cure. The Wassermann reaction should be determined and if positive a course of anti-syphilitic treatment is given (see p. 570).

### Raynaud's Disease

**Definition** A vaso motor disorder affecting the extremities characterised by vascular changes with a tendency to gangrene.



— Normal person after one hour at room temperature of 78° F.

.... Normal person after wrapping in blanket for one hour at room temperature of 78° F.

--- Patient after one hour at room temperature of 78° F.

- - - - Patient after wrapping in blanket for one hour at room temperature of 78° F.

— Patient after two months' Carnation treatment and after one hour at room temperature of 78° F.

- - - - Patient after two months' Carnation treatment and after wrapping in blanket for one hour at room temperature of 78° F.

FIG. 16. SKIN TEMPERATURES IN HEALTH AND IN THROMBO-ANGITIS OBLITERANS.

a cerebral thrombosis. The temperature is usually elevated with each recurrence of thrombosis, and the blood shows a leucocytosis.

**Prognosis** This is usually favourable, even when thrombosis occurs in internal organs, although there is a tendency to recurrence.

**Treatment** The patient must be kept in bed. Plenty of fluids should be taken by mouth and a mixture containing Sod. citrat. gr 30, Syr. aurant. in 20 aqua ad fl. oz. 1 is given every 6 hours. Subsequently the teeth should be X-rayed and any septic focus eradicated. A bacteriological examination of the urine and faeces should also be made, and if pathological organisms are found an autogenous vaccine should be administered.

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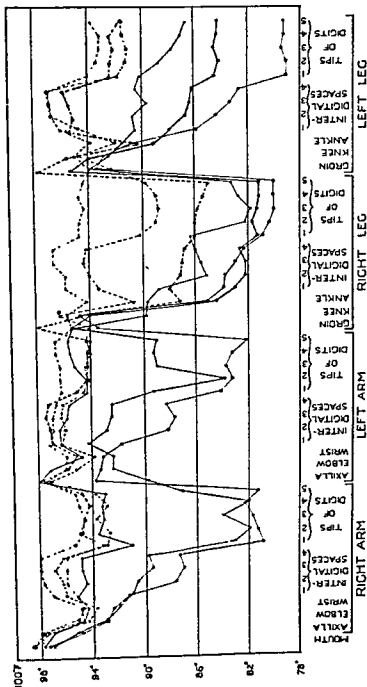


FIG. 16. SKIN TEMPERATURES IN HEALTH AND IN THROMBO-ANGIITIS OBLITERANS.



FIG 17 PHOTOGRAPH TAKEN THE DAY BEFORE AN ATTACK OF ANGIO NEUROTIC EDEMA



FIG. 18 THE SAME PATIENT AS IN FIG 17 DURING AN ATTACK OF ANGIO NEUROTIC EDEMA



**Etiology.** No definite cause is known. It may be associated with a nervous shock or exposure to cold, and rarely with congenital syphilis. It also occurs in workmen who use vibrating tools, such as pneumatic drills.

**Pathology.** No changes are found at autopsy. The circulatory changes are due to vascular spasm, resulting from some local affection of the small arteries rather than to vasoconstriction due to sympathetic impulses.

**Clinical Findings.** The patient is usually a female between the ages of 10 and 30 years. In a mild case (local syncope) the fingers and toes become suddenly pale and dead, and on warming them they ache or throb. *The spasm spreads upwards from the tips of the fingers and toes.* In a moderately severe case (local asphyxia) the patient complains of a tingling sensation in the hands when they are cold. One or more fingers go quite blue or black and there is severe pain. The hands go blue directly, without an initial stage of pallor, if they are held below the level of the heart. The ears or nose may be similarly affected. In a severe case (local gangrene) the pain is almost unbearable, and gangrene, with sloughing, may occur in the fingers, toes, ears or nose. The affected parts may sweat and sensation may be blunted.

**Differential Diagnosis.** Raynaud's disease must be diagnosed from peripheral neuritis, acrocyanosis, erythrocyanosis crurum puellarum frigida, erythromelalgia, thrombo-angiitis obliterans, syringomyelia, and gangrene due to other causes, such as diabetes mellitus, ergotism, etc. There may be vaso-motor changes in the hands or feet with peripheral neuritis, but other signs of neuritis can generally be detected, such as *tenderness along a nerve trunk, or alterations of sensation or of the reflexes.* In acrocyanosis, which usually affects young women, the circulation is poor and the hands and feet are bluish-red and cold, but there are no trophic changes. In syringomyelia there are dissociated sensory changes (see p. 414). In erythrocyanosis crurum puellarum frigida, blue or purplish swellings form after puberty on the backs of the legs and ankles. Indurated nodules may appear in the subcutaneous tissue. The skin is cold and there is a liability to chilblains.

**Course and Complications.** The course is usually progressive for a time; complications include paroxysmal albuminuria. *Hæmoglobinuria* never occurs in true Raynaud's disease.

**Prognosis.** This is variable, but the condition may disappear after several years.

**Treatment.** *Prophylactic.* Vibrating tool workers may have their hands protected by sorbo shock absorbing pads placed in the palms of their gauntlets.

*Curative.* The extremities should be protected from cold. A warm climate is very beneficial. Massage and electrical treatment with galvanism or high frequency should be tried. *Acetylcholine*, in the form of Acécoline 0.1 G. may be injected subcutaneously twice a day for 15 injections. In severe cases affecting the lower limbs, the operation of lumbar sympathectomy may afford a cure. When the upper limbs are affected the results of cervico-dorsal sympathectomy are not so

good possibly because pre ganglionic fibres only are divided by removal of the lumbar ganglia

### Angio-neurotic (Edema

#### (*Quincke's Disease*)

**Definition** A condition characterised by paroxysmal attacks of oedematous swelling of the skin, subcutaneous tissues or mucous membranes

**Etiology** The cause is not known It may be an allergic manifestation or a vaso motor disturbance

**Pathology** The oedema may result from the local effect of toxins on capillaries

**Clinical Findings** The patient is usually a young adult of either sex, and a history is sometimes obtained of similar attacks in other members of the family He complains of localised swellings appearing suddenly in the face, lips, eyelids, mouth, hands or elsewhere An urticarial rash may develop simultaneously The eyelids or lips may *swell and become very tense and painful Thus the eyes may be completely closed* Similar swellings may develop in the tongue or larynx causing urgent dyspnoea The mucous membrane of the alimentary tract may be affected, the patient complaining of acute abdominal pain with vomiting or diarrhoea

**On Examination** The appearance of the patient may be completely altered during the attacks owing to the swelling (see Figs 17 and 18) The cutaneous swellings pit slightly on pressure the skin is generally white but may be a little pink

**Differential Diagnosis** Other varieties of oedema, such as that produced by disease of the heart or kidneys must be excluded The abdominal variety is suggestive of an acute abdominal lesion but there is no fever and a history of previous attacks affecting the skin may afford the clue to the diagnosis

**Course and Complications** Recurrent attacks are to be expected Haemoglobinuria may be a complication

**Prognosis** The patient often outgrows the attacks Death may ensue from oedema of the glottis

**Treatment** *During the attack* An injection of m 8 to 16 of liq adrenal hydrochlor should be given subcutaneously With oedema of the glottis tracheotomy or intubation of the larynx may be necessary to save life *Between the attacks* A search should be made for septic foci in such sites as the teeth, tonsils, antra sinuses, intestines, kidneys, bladder and genital organs Any such focus should be adequately treated The protein cutaneous tests should also be performed The effect of different diets should be determined eliminating such articles as milk eggs or fish

### Milroy's Disease

#### (*Hereditary Trophoedema Non filarial Elephantiasis*)

**Definition** A familial disease characterised by swelling of the legs, arms or face.

**Etiology.** The cause is not known. It is more common in women than in men, and may be noted shortly after birth or not be apparent until after the age of puberty.

**Pathology.** There is thickening of the skin and subcutaneous tissues.

**Clinical Findings.** The patient may say that other members of her family are similarly affected. She complains of heaviness and swelling of the feet and legs. Only one limb may be affected. It is uncommon for the arms or face to be involved.

**On Examination:** The affected limb is usually pale, swollen and pits slightly on pressure.

**Differential Diagnosis.** Other causes of swelling of the extremities must be excluded, such as diseases of the heart or kidneys, varicose veins, obesity, intra-abdominal venous obstruction or lymphatic obstruction due to filarial elephantiasis (see p. 724).

**Course and Complications.** The course is progressive, but it may be interrupted by acute attacks of pain in the limbs accompanied by fever.

**Prognosis.** The disease is neither fatal nor can it often be cured.

**Treatment.** The legs should be bandaged from the feet upwards to prevent the swelling from increasing. Kondoléon's operation is sometimes performed with satisfactory results. Strips of œdematous skin, subcutaneous tissue and deep fascia are removed from both sides of the leg, from the upper end of the thigh to the ankle, with an interval of about 3 inches on each side of the knee.

### Anæsthetics and Cardio-vascular Disease

The physician is often asked to examine the heart of a patient prior to the administration of an anæsthetic for an operation, although the advice given is not always acted upon by the anæsthetist or the surgeon. The ideal procedure would be for every patient to be examined thoroughly according to a definite plan several days before the operation. A formal stethoscopic examination immediately before the operation cannot be too strongly deprecated. Symptoms pointing to myocardial weakness must first be noted, such as undue dyspnœa on exertion, cardiac asthma or precordial pain. If possible the exercise tolerance should be determined. The size of the heart, presence of valvular lesions, the rhythm and the condition of the myocardium are next investigated. Special attention should be paid to the length and tone of the first sound at the apex and to the rhythm. With myocardial weakness there is a tendency for the first sound to be short and of high pitch, and for a tic-tac rhythm to be present. Gallop rhythm, pulsus alternans, auricular fibrillation or flutter, and heart-block are all of serious import. The condition of the arteries and the blood pressure should also be recorded. An electrocardiogram and telerradiogram or orthodiagram should be made in each case, when the condition of the patient permits. Thus with fatty degeneration of the heart there may be no enlargement and no congestive failure, and yet the myocardium is in a parlous state and the surgical risk is great. Statistical evidence

indicates that death under anæsthesia is most to be feared with coronary thrombosis and the risk diminishes progressively with congestive failure, syphilitic aortitis, angina, myocardial degeneration, auricular fibrillation and valvular disease. Valvular disease alone, apart from congestive failure and myocardial weakness, is not a contra indication to operation. A patient who has auricular fibrillation or congestive failure should be digitalised and an operation not performed until his condition has improved. With thyrotoxicosis, however, auricular fibrillation will often only disappear after thyroidectomy has been performed. Such a patient should therefore be operated on after a preliminary course of digitalis and Lugol's solution (liq. iodi aquosus B.P. Add.), although a normal rhythm has not been restored. Gas and oxygen anæsthesia is contra indicated in cases of hypertensive heart disease, as it tends to raise the blood pressure. Spinal anæsthesia, morphine and chloroform are best avoided in low blood pressure, as they are apt to produce a further fall in tension. In every case estimation of the cardio vascular surgical risk is very difficult, but no scientific attempt can be made unless all the requisite clinical data are to hand.

## CHAPTER IV

### THE NERVOUS SYSTEM

**Introductory.** Every patient who is suspected to be suffering from a disorder of the nervous system should be examined in accordance with a definite plan, as detailed later (see p. 283). An elementary knowledge of the anatomy and physiology of the nervous system is essential.

#### Anatomy and Physiology

**The Motor Path from the Cerebral Cortex to the Muscles.** Voluntary muscular movements are initiated by impulses arising in the cerebral cortex, and passing to the mid-brain, pons, medulla or spinal cord. From these lower levels fresh fibres arise and pass direct to the muscles concerned. It is believed that there are three relays of fibres between the cerebral cortex and the muscles, as described below. The motor cortical area lies in the frontal lobe of the brain on each side just anterior to the fissure of Rolando (see Fig. 30). The motor fibres converge and pass through the genu and anterior two-thirds of the posterior limb of the internal capsule of the brain. This is the portion of white matter which lies between the caudate nucleus and optic thalamus on its inner side, and the lenticular nucleus on its outer side. It will be noticed (see Fig. 19) that in the convergent movement the fibres for the leg cross those of the arm and come to lie posteriorly, whereas the face fibres also cross the arm ones to become anterior, the arm fibres being situated centrally. The order of fibres in the internal capsule is therefore, from before backwards, face, arm, leg.

The upper motor neurones emerge from the internal capsule and run through the mid-brain, pons and medulla, near the ventral surface, in a bundle called the pyramid. Certain cranial motor nerves have their cells of origin in this part of the brain. Thus the nucleus of the III nerve lies at the level of the superior corpora quadrigemina (see Fig. 33), and that of the IV nerve in the region of the inferior corpora quadrigemina (see Fig. 34). The motor nucleus of the V nerve is in the middle of the pons, and the VI and VII nuclei in the lower part of the pons. The IX, X, XI and XII nuclei are in the medulla (see Fig. 44). It will be noticed that with the exception of the III and IV nerves, the cranial nerves pass to the same side of the body as that on which their nuclei are situated. Decussation of certain fibres occurs with the III nerve and total decussation in the case of the IV nerve, so that the nucleus of the IV nerve situated on the right side of the brain supplies the left superior oblique muscle, and *vice versa*. There is a bilateral cortical innervation for all the cranial nerves except the XII and the part of the VII nerve which supplies the lower part of the face. As the upper neurones, which constitute the pyramidal tract, pass down through the mid-brain, pons and medulla, they give off

fibres which cross the mid line and end in arborisations in grey matter from which the second relay connects with the dendrites of the motor cranial nerve nuclei

On tracing the pyramidal tract still lower, there is, at the level of the lower part of the medulla a decussation affecting 90% of the fibres. The fibres after decussation are known as the crossed pyramidal tract, and they run down the spinal cord in the lateral regions (see

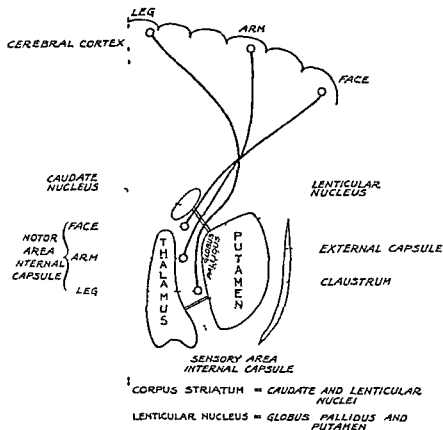


FIG. 19. DIAGRAM SHOWING THE INTERNAL CAPSULE AND CORPUS STRIATUM

Fig. 20) The 10% of fibres which have not decussated form the direct pyramidal tract and pass down the anterior part of the cord near the median fissure as far as the lower cervical or mid thoracic region (see Fig. 20). The fibres of the crossed pyramidal tract are connected with posterior horn cells and then by a second relay with the motor cells in the anterior horn of the grey matter of the spinal cord. These are the cells of origin of the lower motor neurones from which nerve fibres proceed direct to the skeletal muscles. The upper motor neurones in the direct pyramidal tracts cross the mid line in the anterior white commissure of the cord and terminate around posterior horn cells.

They are connected by a second relay with anterior horn cells. There are thus three relays between the cerebral cortex and the muscles, the upper motor neurones, the intermediate neurones, and the lower motor neurones.

**The Extra-pyramidal Motor Tracts.** (The striato-spinal motor system.) These tracts convey involuntary motor impulses from various ganglia in the brain to intermediate relay stations, or to the anterior horn cells of the cord. They are concerned with automatic actions and are an older motor system than is the pyramidal one. They function in infants before the pyramidal system is capable of so doing. The most important of these tracts are:—

1. *The Vestibulo-spinal Tract.* The fibres arise in the lateral

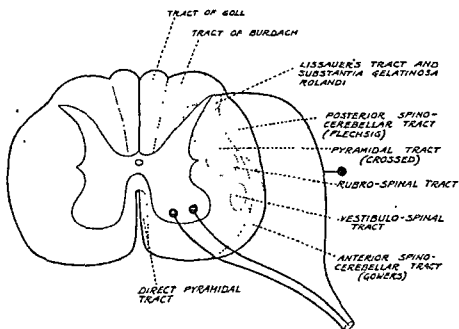


FIG. 20. DIAGRAM OF SECTION OF SPINAL CORD.

vestibular nucleus of the VIII nerve (Deiters' nucleus) in the lower part of the pons and run down the antero-lateral part of the medulla and cord to end around anterior horn cells on the same side of the cord (see Fig. 31).

2. *The Tecto-spinal Tract.* This tract arises in the mid-brain at the level of the superior corpora quadrigemina, and, after crossing in Meynert's or the fountain decussation, it passes down the cord in the anterior longitudinal bundle to end around anterior horn cells. It conveys impulses to the voluntary muscles (eye protection movements) as the result of stimuli from the eyes which have passed to the calcarine fissure and thence to the superior corpora quadrigemina (see Fig. 32).

3. *The Cerebello-rubral Tract.* The fibres arise in the lateral lobe of the cerebellum, and pass by the superior cerebellar peduncle to the red nucleus of the opposite side (see Fig. 31).

**4 The Striato rubral Tract** (The large celled pallidal system) The fibres arise in the globus pallidus of the lenticular nucleus and run to the red nucleus on the same side of the brain. The impulses are concerned with emotional and associated movements.

**5 The Small celled Neo striate System** Fibres pass from the caudate nucleus and the putamen of the lenticular nucleus to the globus pallidus. They exert an inhibitory influence over the motor impulses arising in the large celled pallidal system.

**6 The Rubro-spinal Tract** The fibres arise in the red nucleus, which is situated in the mid brain at the level of the superior corpora quadrigemina. The fibres immediately decussate and pass down the opposite side through the pons and medulla and antero lateral part of the cord to end around anterior horn cells. They convey impulses for group movements (see Fig 31).

**Upper Motor Neurone Lesions** Irritative lesions cause convulsive movements of the voluntary muscles.

Paralytic lesions result in paralysis of voluntary movement with spasticity, increase of deep reflexes, ankle and patellar clonus, and an extensor plantar response. The electrical reactions are normal, and wasting is slight. When the upper motor neurones arising in the cerebral cortex are put out of action, the extra pyramidal motor tracts arising in the basal ganglia may show symptoms of uncontrolled activity, which are known as "release" symptoms. Various involuntary movements then ensue, such as are seen clinically in athetosis following a hemiplegia.

**Extra-pyramidal Motor Neurone Lesions** Lesions of the large celled pallidal system (globus pallidus) as occur in paralysis agitans and progressive lenticular degeneration cause hypertonus of muscles, tremors and disturbance of automatic movements. Lesions of the small celled neo-striate system produce athetoid or choreic movements. Lower motor neurone lesions result in weakness of muscles, wasting, hypotonus, absence of the deep reflexes and a reaction of degeneration.

**The Sensory Path from the Periphery to the Brain** **1 The Spinal Nerves** In the spinal motor nerves about 40% of the fibres are afferent, conveying sensations which are known as proprioceptive or kinæsthetic, from the muscles, tendons, joints and periosteum. Their cells of origin are situated in the posterior root ganglia. The spinal sensory nerves convey impulses of pain, touch and temperature from the skin. These are called exteroceptive impulses. Enteroceptive impulses pass from the viscera by various spinal nerve roots, and connect with cells in the lateral horn of the grey matter of the cord. The following impulses enter the spinal cord by various posterior root fibres (see Fig 21):  
**1** Touch (deep cutaneous), and discrimination of the points of a compass.  
**2** Muscle sense, as evidenced by judging the differences in weight, stereognosis or judging the nature of objects by feeling them, deep pressure pain, tendon sense, joint sense, bone vibration sense and the knowledge of the position of the limb in space.  
**3** and **4** Muscle tonus, equilibrium and co-ordination.  
**5** Cutaneous pain and temperature.  
**6** Localised touch.  
**7** Direct cutaneous—muscle reflex path.



1, 2, 3 and 4 are kinæsthetic sensations, 5 and 6 are skin sensations.

2. *The Central Path* (see Figs. 21, 22). *Impulses 1 and 2.* The fibres conveying deep touch, discrimination of compass points, muscle, tendon, joint and bone sense pass up the cord on the same side in the posterior column of Burdach. As they ascend they are pushed centrally

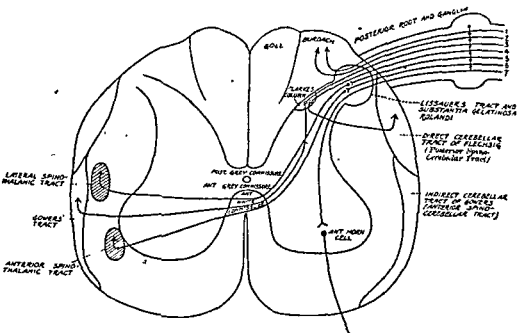


FIG. 21. DIAGRAM SHOWING FIBRES OF A POSTERIOR NERVE-ROOT ENTERING THE CORD.

*Posterior Root Tracts—*

1. Touch (deep cutaneous). Discrimination of points of compass.
2. Muscle sense. Joint sense. Tendon sense. Bone vibration sense. Pressure-pain of muscles.
- 3 and 4. Muscle tonus. Equilibrium. Co-ordination.
5. Pain. Temperature.
6. Localised touch.
7. Direct cutaneous muscle reflex.

*Posterior columns: Muscle sense*, such as sense of position of a limb, judging weights, pressure pain.

*Stereognostic sense*, a combination of touch and joint sense.

*Joint sense* (position of a joint).

*Bone vibration sense.*

*Tactile*, discrimination of two points. Sense of position of skin when pulled out.

*Lateral Spino-thalamic Tract.* Pain and temperature.

*Anterior Spino-thalamic Tract.* Highly localised touch.

*Spino-cerebellar Tracts* (Gowers and direct cerebellar). Muscle tonus.

by new fibres coming in and so they constitute the column of Goll. The fibres terminate in the gracile and cuneate nuclei in the lower part of the medulla. The gracile nucleus is the relay station for fibres from the leg, and the cuneate nucleus for the fibres from the arm. The Th. 3-12 nerves do not appear to have any connection with the gracile or cuneate nuclei. A second relay of fibres now begins which decussates (decussation of the fillet) and then ascends the pons and mid-brain as

the mesial fillet, to the antero lateral nucleus of the optic thalamus. From the optic thalamus some impulses pass by a third relay to the sensory cerebral cortex in the posterior central gyrus behind the Rolandic fissure. Other fibres pass from the gracile and cuneate nuclei to the cerebellum by the superficial and deep arcuate fibres which run into the inferior cerebellar peduncle (see Fig 31).

*Impulses 3 and 4* Muscle tonus sensations and impulses for maintaining equilibrium relay first in Clarke's column near the posterior

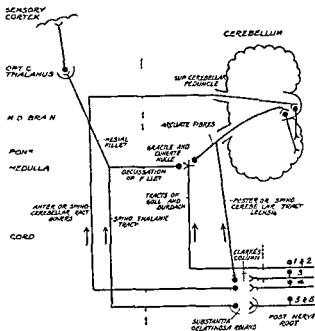


FIG 22 DIAGRAM SHOWING THE SENSORY PATH

*Fibres in Posterior Root—*

1 & 2 Kinesthetic

3 & 4. Equilibrium

5 & 6 Pain Temperature and some touch sensations

horn of grey matter. The second relay of fibres conveys the impulses either to the direct cerebellar tract of Flechsig (posterior spino cerebellar tract) on the same side, or cross the mid line in the grey or the anterior white commissure to the indirect cerebellar tract of Gowers (anterior spino cerebellar tract) on the other side. The direct cerebellar tract conveys the impulses to the cerebellum on the same side by the inferior cerebellar peduncle, whereas the indirect cerebellar tract decussates higher up (see p 351) and passes to the cerebellum by the superior cerebellar peduncle, on the same side as that on which the impulses entered the cord.

*Impulses 5 and 6* Sensations of pain and temperature enter the cord

by the posterior root and relay in the substantia gelatinosa Rolandi. The second relay conveys the impulses to the opposite side of the cord through the anterior white commissure and they then ascend in the lateral part of the spino-thalamic tract. This joins the mesial fillet and runs to the antero-lateral nucleus of the thalamus. Some of the impulses are then carried by a third relay to the sensory part of the cerebral cortex (the post-central gyrus, the superior parietal lobule, the supramarginal and angular gyri), and others pass to the mesial thalamic nucleus (the essential thalamic organ) where they give rise to sensations of pleasure and pain. The fibres conveying the highly localised sense of touch also relay in the substantia gelatinosa Rolandi. The second relay crosses the mid-line in the anterior white commissure and ascends in the anterior part of the spino-thalamic tract. These fibres join the mesial fillet, and terminate in the optic thalamus. A final relay conveys the impulses to the sensory part of the cerebral cortex.

The Cranial Nerves (see p. 356). The sensory fibres from the V, VII, IX and X nerves also join the fillet.

The Cerebellar Connections (see p. 351).

#### Nervous Disease Case Sheet

Name. Age. Sex. M.S.W.

Family history of nervous diseases.

Previous history.

History of present illness. Date of onset.

Mode of onset.

General condition. Pulse. Temperature.

Respirations. Urine. Blood (cell count and W.R.). Cerebration.

Speech.

Cranial Nerves. I.

II.

III. IV. VI.

V. Motor. Sensory.

VII. Motor. Sensory.

VIII.

IX. X. and accessory part of XI. Motor. Sensory.

XI. Spinal part.

XII.

Spinal Motor Nerves. (a) Power of muscles.

(b) Wasting or hypertrophy of muscles.

(c) Tone of muscles.

(d) Tremors, fibrillations, spasms or convulsions.

Spinal Sensory Nerves. (a) Tactile discrimination (compass test).

(b) Muscle sense.

(c) Stereognostic sense.

(d) Muscle and tendon pressure sense.

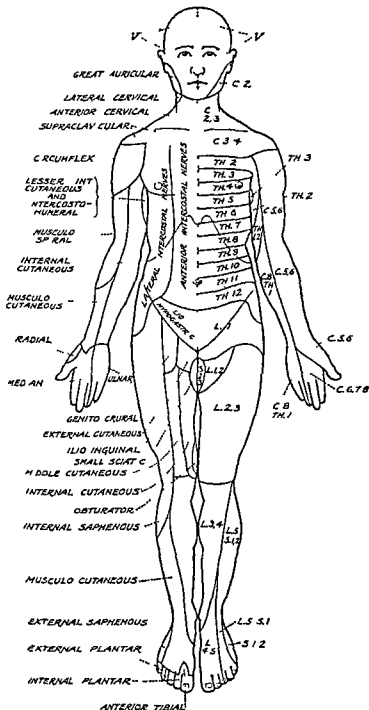


FIG 23A. THE AREAS OF SKIN SUPPLIED BY THE SPINAL NERVES ARE SHOWN IN THE LEFT HALF OF THE FIGURE, AND THE SKIN AREAS SUPPLIED BY THE PERIPHERAL NERVES ARE INDICATED IN THE RIGHT HALF OF THE FIGURE. (After Cunningham.)

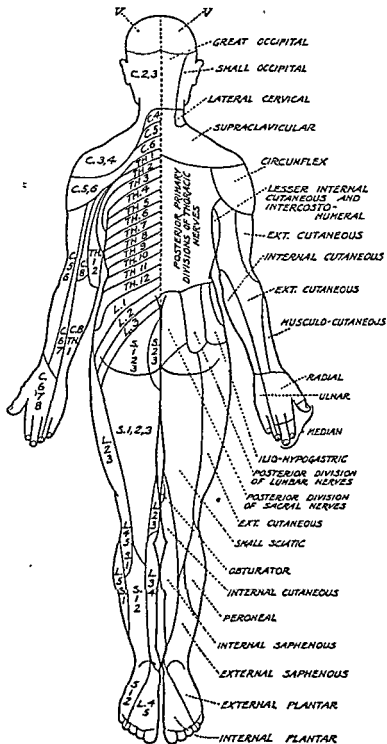


FIG. 23B. THE AREAS OF SKIN SUPPLIED BY THE SPINAL NERVES ARE SHOWN IN THE LEFT HALF OF THE FIGURE, AND THE SKIN AREAS SUPPLIED BY THE PERIPHERAL NERVES ARE INDICATED IN THE RIGHT HALF OF THE FIGURE. (After Cunningham.)

- (e) Joint sense.
- (f) Vibration sense.
- (g) Pain, temperature and light touch.
- (h) Subjective sensation.

**The Reflexes.** (a) *Superficial.*

- 1 Conjunctival (V).
- 2 Palatal (IX and X).
- 3 Pharyngeal (IX).
- 4 Epigastric (Th. 7-9)
- 5 Abdominal (Th 9-12).
- 6 Cremasteric (L 1 and 2).
- 7 Plantar (S 1 and 2).

(b) *Deep.*

- 1 Pupil (II and III).
- 2. Jaw jerk (V)
- 3 Biceps (C 5 and 6)
- 4. Triceps (C 6 and 7).
- 5 Supinator (C 7 and 8)
- 6 Knee jerk and clonus (L 2-4)
- 7. Ankle jerk and clonus (S 1 and 2).

(c) *Visceral.*

- 1 The bladder
- 2. The rectum

**Co-ordination** (a) The upper limbs

(b) The lower limbs.

**Trophic Changes.** (a) Skin

(b) Bones and joints.

**Gait.**

**Electrical Reactions**

**Lumbar Puncture.**

**Site of Lesion.**

*Additional notes on the examination of the nervous system :—*The chief segmental nerve supply for some of the important muscles is as follows C 4. Diaphragm. Trapezius C 5 Supraspinatus. Infraspinatus Biceps. Deltoid C 6 Pronators of forearm Latissimus dorsi. C 7. Triceps Extensors of wrist and fingers C 8. Flexors of wrist and fingers. Th. 1. Intrinsic muscles of the hand. Th. 2-10 Intercostals. Th. 7-12. Abdominal wall muscles. L 1 Quadratus lumborum. L 3. Adductors of thigh. Iliopsoas L 4. Extensors of knee and abductors of thigh. L 5. Flexors of knee. S 1. Glutei and calf muscles. S 2. Anterior tibial muscles. Peronei. Intrinsic muscles of foot. S 3 and 4.

Pelvic muscles. The cutaneous peripheral nerve and nerve root areas are illustrated in Fig. 23.

**Co-ordination.** (a) Upper limbs. This is tested by the ability to pick up small objects, and to touch the tip of the nose, with the eyes shut.

(b) Lower limbs. Romberg's sign. The patient cannot stand with the feet together and eyes closed. Co-ordination is tested also by asking the patient while lying down to touch with his toe an object held near it, or to touch his knee with the opposite heel.

(c) Past-pointing. This test is described on p. 379.

**Trophic Changes.** *Skin*, e.g., glossy skin, perforating ulcers, painless whitlows, herpes zoster, bed sores, leucoderma, and increased sweating. *Bones and joints*, e.g., facial hemiatrophy or hemihypertrophy, inequality of the two halves of the body, arthropathies.

**The Gait.** Spastic, ataxic, cerebellar, scissors, high stepping, reeling, festinating, hysterical, etc.

**The Electrical Reactions.** The stimulus is applied at the motor point where the nerve enters the muscle. Normally muscle directly stimulated responds to the make and break of a galvanic current, but not to a faradic stimulation. If the muscle is stimulated through the nerve at the motor point, it normally responds to faradisation and also to the make and break of the galvanic current.

**Reaction of Degeneration.** The muscle, when stimulated at the motor point, does not respond to faradic stimulation owing to nerve degeneration, and the response to galvanism is modified so that it is sluggish, or the anodal closure contraction is greater than the kathodal closure contraction, i.e., A.C.C. 7 K.C.C. Normally the reverse is the case.

**Lumbar Puncture.** Normal cerebro-spinal fluid: Pressure 150 mm. H<sub>2</sub>O. Cells 1 to 5 mononuclears per cmm. Protein 0.015 to 0.03%. Urea and non-protein nitrogen 15 to 30 mg., chlorides 700 to 750 mg., sugar 70 to 100 mg. per 100 c.c.

**Lipiodol Examination.** By injecting Lipiodol into the cisterna magna or into the theca by lumbar puncture the site of the spinal cord obstruction can be visualised by X-ray examinations.

In the diagnosis and localisation of intracranial tumours certain other investigations may be required. These include:—

**Radiography.** The skull must be X-rayed from several angles. By this means information is obtained as regards the size and shape of the skull, the presence of bossing, the bony texture, the presence of fracture, the state of the sutures, the presence of erosion of the dorsum sellæ or clinoid processes, the vascular markings, the presence of abnormal or normal calcifications and the size of the optic foramina, etc.

**Ventriculography** (see p. 318).

**Encephalography** (see p. 318).

**Angiography.** Thorotrast (8 to 10 mls) are injected into the internal carotid artery, and X-ray films of the skull are taken during the injection and 3 seconds later. This method, which is not without danger owing to the toxic nature of thorotrast, assists in the diagnosis of aneurysms and malignant gliomas.

**Electro encephalography** Changes in electric potential arising in the cerebral cortex can be demonstrated with electrodes applied to the scalp. The normal electro encephalogram shows characteristically

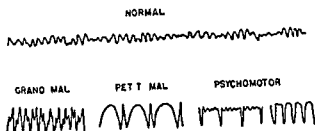


FIG. 24. NORMAL ELECTRO ENCEPHALGRAM AND TYPES OF WAVES IN EPILEPSY  
LENNOX *Jour Amer Med Assoc*

small alpha waves. With a cerebral tumour or abscess and in epilepsy abnormal waves may be seen (see Fig. 24 and pp. 311-331).

## MENINGITIS

**Definition** Inflammation of the meninges

**Varieties** 1 *Pachymeningitis* is inflammation of the dura mater. This is often secondary to disease or injury of the bone, such as spinal caries or a fractured skull. Haemorrhagic pachymeningitis is met with in general paralysis of the insane (see p. 392). In chronic alcoholism and senile dementia it probably results from slight injuries to the skull not noticed by the patient. Pachymeningitis cervicalis hypertrophica may be due to syphilis (see p. 387) and less frequently to tuberculosis.

2 *Leptomeningitis* implies inflammation of the pia arachnoid. There is often also ependymitis or inflammation of the lining membrane of the cerebral ventricles. Leptomeningitis is usually due to invasion of the cerebro spinal fluid by micro organisms such as the *Mycobacterium tuberculosis* (*B. tuberculosis*), the *Neisseria meningitidis* (meningococcus), *Diplococcus pneumoniae* (pneumococcus), the streptococcus, the staphylococcus, the *Neisseria gonorrhoea* (gonococcus), the *Bacterium typhosum* (*B. typhosus*), the *Bacterium commune* (*B. coli*), the *Treponema pallidum* and possibly the *Haemophilus influenzae* and the *Diplococcus rheumaticus*. Aseptic meningitis and serous meningitis are also described.

3 *Arachnoiditis* is inflammation of the layers of the arachnoid, with cyst formation.

### Tuberculous Meningitis

#### (Basilar Meningitis)

**Etiology** The disease is caused by the *Mycobacterium tuberculosis* (*B. tuberculosis*) 75% being of the human and 25% of the bovine type.

**Predisposing causes** 1 Age. Usually children between the ages of 1 and 6. Adults may be affected. 2 Sex. Equal incidence.

**Pathology** The organisms are usually carried to the meninges by the blood stream from a tuberculous gland in the chest or abdomen. In



some cases the meningitis is secondary to tuberculous lesions in the lungs, bones, joints, skin or genito-urinary organs. Rarely the meningitis is due to a direct spread from a tuberculous focus in the brain. An operation on a tuberculous joint may cause a generalisation of the tuberculous infection with resultant meningitis. At autopsy there is inflammation of the pia at the base of the brain, especially in the interpeduncular space, the Sylvian fissure, and optic chiasma. Minute tubercles may be seen on the pia or on the branches of the middle cerebral, anterior and posterior perforating arteries. The cerebral convolutions are somewhat flattened, the brain substance softened, and the ventricles contain an excess of cerebro-spinal fluid producing a moderate degree of hydrocephalus. Minute tubercles may be found in other parts of the body, such as the lungs, liver and spleen, if a condition of miliary tuberculosis exists.

**Clinical Findings.** The patient is often a child of about 3 years of age. In some cases there is a history of a blow on the head some weeks previously or of a recent attack of measles or of whooping-cough. Often the patient has been apparently in good health, but for a few weeks previously has become listless and irritable, with headache, loss of appetite, slight temperature and loss of weight. The first symptom noted may be a convulsion. In adults tuberculous meningitis may be a terminal complication of pulmonary tuberculosis.

**On Examination:** In the early stages the patient is usually rather irritable, restless and does not like being examined, drawing up the bed-clothes over himself. He may cry out from time to time ("hydrocephalic cry") and is easily startled by noises. He lies on his side with the knees drawn up. Twitching of various muscles may be seen and there may be photophobia. The only signs of an organic lesion which may be found are strabismus, due usually to weakness of the external rectus muscle, ptosis and some rigidity of the neck and back muscles. The pupils are rather small and the child may grind his teeth. Flexion of the hip with the knee extended is resisted (Kernig's sign) and similarly extension of the shoulder with the elbow extended may cause pain (Bikele's sign). Flexion of the neck causes flexion of one or both hips and knees (Brudzinski's sign). The temperature is raised to 101° or 103° F., and the pulse is frequent, such as 100 to 120. The abdomen is rather scaphoid, the bowels are constipated and vomiting occurs apart from taking food (cerebral vomiting). The deep reflexes are usually exaggerated. This is the *stage of irritation*.

A few days later a *stage of compression* may be reached. The patient is now definitely drowsy or comatose, but localised or general muscular convulsions may be seen, with irregular facial contractions. There is more marked head retraction but the back is rarely arched. The pupils are dilated and often unequal, and the patient may complain of difficulty in reading. In infants the anterior fontanelle may bulge. Optic neuritis and less often choroidal tubercles may be found on ophthalmoscopic examination. The pulse is slower and premature systoles may be noted, the temperature is often lower, about 99° in the evenings. Vomiting usually ceases during this stage. Later still, a *paralytic stage*

may be reached with incontinence of faeces and urine and a low temperature. Vomiting may now recur. The pulse tends to be more frequent and is often very rapid shortly before the end. Just before death the temperature may rise to 106° F or higher. The pupils are dilated and do not react to light. The anterior fontanelle now becomes flat. The child lies on his back with the legs extended. The abdominal and deep reflexes are abolished. In many cases it is not possible to differentiate these stages. The cerebro spinal fluid. The fluid is under pressure, clear or slightly turbid. On standing a fine coagulum often forms. Tubercle bacilli may be found in the majority of cases in the coagulum. Lymphocytes are present in excess, up to 400 or more per cmm. In the early stages however, 50 or 60% of the cells may be

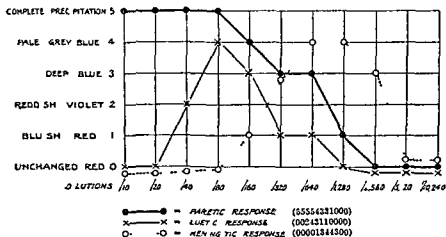


FIG. 2. LANGE COLLOIDAL GOLD CURVES

polymorphonuclears. There is an excess of protein and the Nonne Apelt test for globulin is positive. Sugar is diminished to 15 or 20 mg per 100 c.c. and the chlorides are reduced to 600 or 650 mg per 100 c.c. The Lange test shows a meningitic curve (see Fig. 25). The blood. A leucocytosis of about 12,000 per cmm may be present.

**Differential Diagnosis.** In the early stages the illness may be mistaken for influenza or gastritis. Thus headache, vomiting, drowsiness, fever and convulsions may all occur in gastro-enteritis. Apical pneumonia in a child may at the onset closely simulate meningitis. In meningism there are symptoms and signs of meningitis, although the results of lumbar puncture show no abnormality. The prolonged fever may suggest typhoid fever. The nervous symptoms may be confused with polioencephalitis or with encephalitis lethargica. A cerebral tumour or abscess may be confused with tuberculous meningitis, but in the former optic neuritis is usually severe and cranial nerve palsies are not so apparent. If the patient is comatose, other causes of coma such as diabetes mellitus and uræmia must be excluded. The diagnosis of meningitis is established by the results of lumbar puncture, which also differentiate other varieties of meningitis.

stage may be followed about a week later by convalescence, the headache and rigidity passing off and the temperature falling to normal, or there may be a more prolonged chronic stage.

*The Chronic Stage:* The chief clinical features are marked wasting despite a good appetite, vomiting, rigidity, muscular twitchings, hydrocephalus, dementia, convulsions, strabismus, nerve deafness, ptosis and at times facial paralysis. There is often incontinence of urine and faeces. The cerebro-spinal fluid at this stage may show the characteristics of Froin's syndrome, if spinal block develops above. The fluid obtained by lumbar puncture below the block is clear and faintly yellow (xanthochromia). Such a stage may last for 2 to 3 months and the patient may then die or gradually recover.

*Varieties:* 1. Chronic meningococcal septicaemia without meningitis: The chief clinical features are recurrent attacks of fever, rigors, and small crops of petechiae seen especially on the lower parts of the legs, the dorsum of the feet, and the hands. The condition can only be diagnosed by blood culture. 2. Fulminating: Death may occur in a few hours or days, the mind remaining clear in the suprarenal type, and the patient being stuporose and later comatose in the encephalitic type. 3. Mild or ambulatory: Characterised by short but severe occipital headache, slight pyrexia and perhaps labial herpes. 4. Posterior basic meningitis of infants: This may be epidemic or sporadic. The onset may be insidious, but is often sudden with convulsions and vomiting followed by head retraction, with opisthotonus. The disease soon passes into the chronic stage with wasting, hydrocephalus and blindness due to cortical lesions. The hands and feet may show the characteristic attitude of tetany. Lumbar puncture usually is "dry" owing to blocking of the foramina of Magendie and Luschka. Puncture of the ventricles shows turbid fluid containing meningococci. The disease is usually fatal in 1 or 2 months, but recovery may occur, the child being often mentally deficient or blind.

*Differential Diagnosis.* Cerebro-spinal fever must be diagnosed from other causes of convulsions in infants, such as rickets, gastro-intestinal disturbances, or the onset of the infectious fevers.

If there are no meningeal symptoms other causes of continuous temperature must be excluded, such as typhoid fever, influenza or pneumonia. The presence of a purpuric rash and arthritic symptoms may suggest purpura rheumatica, and a sudden onset with rash may be mistaken for typhus fever. When meningeal symptoms are present it must be diagnosed from meningism, other varieties of meningitis, polioencephalitis, encephalitis lethargica and cerebral tumour. Other causes of coma must also be excluded. Examination of the cerebro-spinal fluid establishes the diagnosis.

*Course and Complications.* Recrudescence of symptoms during the febrile period which was formerly common, is now very rare if adequate sulphonamide treatment is given. Relapses after an apyrexial interval are rare.

Complications include blindness, which may be central or due to suppuration in the eye, nerve deafness and hydrocephalus, hemiplegia,

monoplegia, paraplegia, spastic ataxia, bronchopneumonia and arthritis with or without effusion. Acute hæmorrhagic nephritis may be the initial symptom. Sequelæ include headaches, mental instability and pains in the back.

**Prognosis.** This has been much improved by chemotherapy, the mortality being lowered to about 5%.

**Treatment. Prophylactic.** In an epidemic, especially amongst troops, the beds should be spaced out to at least 36 inches. Tents should not be overcrowded and adequate ventilation ensured. Carriers are so numerous that they cannot be dealt with effectively. Gargling, spraying or other local treatments to the nose and throat are useless and may be harmful.

**Isolation Period.** The patient should be isolated until he has recovered, and the swab from the naso-pharynx shows no meningococci.

**Curative.** The patient should be put to bed in a well-ventilated room. The skin is tepid sponged twice a day and all pressure points protected by air rings. A water bed may be necessary. Diet. Nasal feeding may be required, but the patient can usually swallow fluids. The bowels should be kept open daily with laxatives such as cascara sagrada. Enemata may be necessary. The abdomen should always be examined to see if there is retention of urine and, if so, a catheter used. **Drugs.** For the headache, insomnia and pains, aspirin gr 5 to 10 t d s, bromides gr 10 t d s, paraldehyde m 120 to 240 at night or an injection of morphin sulph gr  $\frac{1}{4}$  or hyoscin hydrobrom gr 1/200 may be necessary. Ice may be applied to the shaved head and hot baths used to ease the pain in the back.

Lumbar puncture relieves headache and tends to prevent hydrocephalus. It is required for 2 to 3 days in a few cases to relieve pressure symptoms. Frequently it is only necessary at the beginning and end of the treatment in order to establish the diagnosis and to ascertain that the fluid has returned to normal. Sulphanilamide, Sulphapyridine (M. & B. 693) and Sulphathiazole (M. & B. 760) have replaced the use of meningococcus antitoxin, except in very acute cases when they are used together. All these sulphonamides give equally good results but Sulphathiazole is the least toxic. The daily dose in accordance with the age of the patient, is 0 to 2 years, 3 G, 2 to 5 years, 4.5 G, 5 to 10 years, 6 G, 10 to 15 years, 7.5 G, over 15 years 9 G. The daily amount is divided into six equal portions and given every 4 hours by day and night the patient being awakened if necessary. This dosage is maintained for 2 to 3 days then reduced to  $\frac{2}{3}$  for 2 days and to  $\frac{1}{3}$  for 2 days. The drug has little effect after 9 days, and should then be discontinued. The total amount given to an adult is between 45 and 55 G. With adults the first 2 doses may be increased to a maximum of 2 G, but each subsequent dose should not exceed 1.5 G. In very acute cases Sulphapyridine or Sulphathiazole should be given intravenously for the first 2 doses. For an adult 20 or 40 mls of a 5% solution of the sodium salt are injected intravenously, and for a young child or infant 10 mls. The injection may be made into the external jugular vein in infants. The drug should not be given intrathecally.

or intramuscularly. Subsequently the drug is given crushed and suspended in water by mouth or by nasal or pharyngeal tube if the patient cannot swallow. Sufficient fluid must be given by mouth, or parenterally, using normal saline, to prevent or relieve dehydration. In infants the saline may be injected intra-peritoneally. If antitoxin is required, 60 mls for a child, and 120 mls for an adult diluted with an equal volume of warm normal saline are injected intravenously.

### Septic Meningitis

#### *(Pyogenic or Suppurative Meningitis)*

**Etiology.** Septic meningitis may be due to infection with the streptococcus, staphylococcus, the *Neisseria gonorrhœa* (gonococcus), or possibly the *Hæmophilus influenzae* or coliform organisms such as the *Bacterium alkaligenes*. It may follow a direct spread of infection from otitis media, cranial sinusitis, mastoiditis or a cerebral abscess. In other cases it is secondary to septicæmia.

**Pathology.** A purulent infiltration of the meninges covers the vertex, and less often the base of the brain. The ventricles of the brain may be distended.

**Clinical Findings.** The symptoms and signs resemble those described above for other forms of meningitis, such as tuberculous or meningococcal. Thus there is headache, vomiting and usually constipation.

**On Examination:** The temperature is raised to 101° F. or higher, the pulse being about 90 or 100, and often irregular owing to premature systoles. There is irritability and some rigidity of the neck and back muscles. There may also be strabismus and unequal pupils. The patient is often delirious and rigors may occur. The blood: There is a leucocytosis of about 12,000 to 20,000 per c.mm. The cerebro-spinal fluid: This is turbid and under increased tension. There is an excess of leucocytes and the causative organism is present. The protein is increased, but the sugar content is normal.

**Differential Diagnosis.** The presence of meningitis and its type is determined by the results of lumbar puncture.

**Course and Complications.** The disease is rapidly progressive.

**Prognosis.** Death often occurs in 2 to 3 days, but in some cases recovery has been recorded, and the prognosis has been improved by chemotherapy.

**Treatment.** The general treatment is as described above for tuberculous and meningococcal meningitis (see pp. 291, 294). The appropriate sulphonamide preparation should be administered; Sulphanilamide for streptococcal and Sulphathiazole for staphylococcal and gonococcal infections. The method is described on p. 291. Lumbar puncture should be performed to relieve headache.

### Pneumococcal Meningitis

**Etiology.** The *Diplococcus pneumoniae* (pneumococcus) is the causative organism. A primary form is described in which the meninges are first affected, but meningitis is usually secondary to pneumonia,

empyema or pericarditis, the organisms being carried in the blood stream. Direct spread may occur from a pneumococcal otitis media.

**Clinical Findings.** The clinical picture closely resembles that of septic meningitis (see p. 295). There is headache, vomiting, constipation, fever, neck rigidity and often convulsions, with a terminal stage of coma. The cerebro spinal fluid is turbid and under increased pressure. An excess of leucocytes is present, with pneumococci. The protein is increased, but the sugar content is normal or slightly reduced.

**Differential Diagnosis.** The diagnosis is established by the results of lumbar puncture.

**Course and Complications.** The disease is usually quickly progressive.

**Prognosis.** Death may occur in 24 hours or be delayed for a few days. The prognosis has been improved by sulphonamide treatment.

**Treatment.** Sulphapyridine (M & B. 693) has replaced the use of serum. The initial dose should be high and the subsequent doses sufficient to maintain a concentration of 10 to 15 mg per 100 c.c. in the cerebro spinal fluid. Very young infants are given 1 to 3 G., and older children and adults 6 to 12 G. by mouth, as an initial dose. This is followed by a quarter of the initial dose every six hours until the patient seems entirely well clinically, and several successive cultures of the cerebro spinal fluid are sterile. This dose should not be reduced until the temperature has been normal for a week, and half the amount is then given for several days. If the patient cannot swallow, the drug can be administered through a nasal tube. In all cases of pneumococcal meningitis an intravenous injection of a 5% solution of Sodium Sulphapyridine is also given every six hours, the first dose being on the basis of 0.1 G. per kg. body weight, and subsequent doses of 0.03 G. per kg. body weight until two successive cultures of the cerebro-spinal fluid are sterile. The general treatment is as described for tuberculous or meningococcal meningitis (see pp. 291, 294).

### Acute Aseptic Meningitis

#### (Acute Benign or Lymphocytic Meningitis)

**Definition.** A meningitis of acute onset with a favourable course, in which the cerebro-spinal fluid is sterile.

**Etiology.** The cause is unknown, but a virus infection has been postulated in some cases. The condition is probably allied to acute idiopathic benign serous meningitis.

**Clinical Findings.** The patient may be a child or adult. He is suddenly taken ill with headache (usually occipital), vomiting, stiffness of the neck, pains in the neck and limbs, and insomnia. The mind remains clear.

**On Examination.** There is some rigidity of the neck and back, and Kernig's and Brudzinski's signs are present. There may be nystagmus and a squint. The temperature is raised to about 100° or 101° F., and the pulse is about 80 to 90. Ophthalmoscopic examination often reveals swelling of the disc (papilloedema). At times a transient facial palsy may be observed. The cerebro-spinal fluid. This is under increased

pressure, clear or slightly turbid. There is an increase of cells, usually of lymphocytes. In some cases the polymorphonuclear cells are first increased, and later in the illness there is an excess of lymphocytes. The protein may be increased, but the sugar and chloride content are normal. No organisms are present.

**Differential Diagnosis.** The diagnosis is established by the course of the disease and the examination of the cerebro-spinal fluid. It is very liable to be mistaken for tuberculous meningitis until the cerebro-spinal fluid has been examined.

**Course and Complications.** The disease is of short duration, and there are no complications.

**Prognosis.** The patient usually recovers in about 7 to 10 days.

**Treatment.** No special treatment is required beyond a lumbar puncture to establish the diagnosis.

### Acute Serous Meningitis

**Definition.** A meningitis in which the cerebro-spinal fluid is under increased tension, but shows little or no other changes in its composition.

**Etiology.** Serous meningitis may occur without any discoverable cause. The cases are probably the same as acute aseptic meningitis. In other cases it is a complication of otitis media, cerebral abscess, chronic alcoholism ("wet brain," see p. 743), or of encephalitis lethargica.

**Clinical Findings.** The clinical picture may simulate very closely that of tuberculous meningitis. There is lethargy, head retraction, vomiting, headache, fever, Kernig's sign, and papilloedema. The cerebro-spinal fluid is under increased tension, quite clear, and shows no excess of cells, a normal protein and sugar content, and no organisms. In some cases an excess of lymphocytes or of polymorphonuclear cells is found in the fluid.

**Differential Diagnosis.** The diagnosis is established by the results of lumbar puncture.

**Course and Complications.** In the cases for which no cause is found the disease usually pursues a short and favourable course.

**Prognosis.** This is good, apart from cases due to chronic alcoholism.

**Treatment.** There is no special treatment, beyond the nursing required for any case of meningitis, and lumbar puncture.

### Meningism

This is a condition in which there are signs and symptoms of meningeal irritation, but the cerebro-spinal fluid is normal and is not under increased tension. It is probably due to the presence of toxins in the cerebro-spinal fluid. It may occur in apical pneumonia, especially in children.

### Arachnoiditis

#### (*Meningitis Serosa Circumscripta*)

Arachnoiditis may result from trauma or infection, or it may be associated with diseases such as disseminated sclerosis, tabes dorsalis,

syringomyelia, and cerebral tumours, or it may occur as a sequela of leptomeningitis. Cerebral arachnoiditis is often situated in the region of the cortex, the optic chiasma or in the posterior fossa. The symptoms and signs resemble those of cerebral tumour, as the arachnoiditis is characterised by cyst formation. Primary optic atrophy may result from chiasmal arachnoiditis. *Otic hydrocephalus* occurring in association with middle ear infection is closely allied to arachnoiditis. It is suggested that infection of the lateral sinus leads to retrograde thrombosis of the superior longitudinal sinus. Absorption of the cerebrospinal fluid through the arachnoid villi is thereby blocked and hydrocephalus results. Treatment consists in repeated lumbar puncture. Spinal arachnoiditis gives rise to root pains followed by varying degrees of paraplegia. At operation a gush of fluid escapes from the arachnoid cyst, the arachnoid itself showing very slight signs of inflammation.

## THE CEREBRUM

### Intracranial Aneurysms

**Pathology.** The following varieties are described and the order of frequency of their occurrence is 1 *Arteriosclerotic*. Degeneration occurs in the media. 2 *Congenital*. These are thought to be due to weak spots in the muscle coat and are met with especially on the base of the brain at the junction of the anterior cerebral and anterior communicating arteries. They are said to resemble a berry hanging from a stalk. From the clinical standpoint these are of the greatest importance and by some authorities are considered to occur most frequently. 3 *Infective embolic or mycotic*. These result from adhesion of an infected embolus to the intima, and may occur in infective endocarditis or septicæmia. There is a tendency to rapid formation and rupture. Mycotic aneurysms are often found on the middle cerebral artery and occur chiefly in young people. Acute polyarteritis nodosa is another cause, the outer coat of the artery being first affected. 4 *Syphilitic*. The basilar artery is chiefly affected. Some authorities state that this kind of aneurysm does not occur. 5 *Traumatic*. This is usually of the arterio venous type, and situated between the internal carotid artery and the cavernous sinus.

The aneurysms vary in size from that of a pin's head to a fist, the average size being that of a pea. They are usually situated near the base of the brain but may be deep in a hemisphere. The middle cerebral and the basilar arteries are those most commonly affected. Rupture occurs in about 50% of all cases.

**Clinical Findings.** *Before rupture*. There are often no signs or symptoms before rupture occurs, but in some cases there is headache (usually homolateral), irritability, giddiness, tinnitus and nausea. Papilloedema may be present and a localised systolic murmur is sometimes heard on listening to the skull over the aneurysm, especially in the arterio venous type. Variability of signs from time to time is a feature of unruptured intracranial aneurysms. *Other localising signs*



may be present which vary with the artery affected: *Internal carotid*. This results in pressure on the III, IV and VI and upper division of V nerves, with partial or complete internal and external ophthalmoplegia and pain or sensory loss over the forehead and eye. Pressure further forward on the optic path may result in hemianopia and optic atrophy. Non-pulsating exophthalmos may also occur. *Middle cerebral*. There may be Jacksonian fits and hemiplegia or monoplegia. *Anterior cerebral and anterior communicating*. The signs may resemble those of a frontal lobe tumour (see p. 311). *Vertebral*. The syndrome produced may resemble that of thrombosis of the posterior inferior cerebellar artery (see p. 353). *Basilar*. The III, V, VI, VII or VIII nerves may be affected, or there may be pressure on the pituitary. *Arterio-venous aneurysm* of the internal carotid artery and cavernous sinus. This is a cause of pulsating exophthalmos.

*At rupture*. When rupture occurs the symptoms are usually those of subarachnoid hæmorrhage (see p. 302), but at times the hæmorrhage is intracerebral.

**Differential Diagnosis.** Intracranial aneurysms are not usually diagnosed before rupture, but should be thought of when there are symptoms of a cerebral tumour or of involvement of the III, IV, V and VI nerves. When rupture occurs and blood is found in the cerebro-spinal fluid other causes of cerebral hæmorrhage have to be considered, such as hæmorrhage into a glioma. The skull should be X-rayed, for in some instances the calcified aneurysm is revealed. The shadow may be as large as a walnut. Albl describes an almost closed circle shadow, but only a portion of the arc, or several arcs of circles may be shown.

**Prognosis.** Death occurs in about half the cases which bleed.

**Treatment.** Rest in bed is essential, and in some cases a sleepy state may be induced for periods up to 2 months by the administration of hypnotics such as phenobarbitone in adequate doses. Thus phenobarbitone gr. 1 t.d.s. may be required. Lumbar puncture may be advisable (see Subarachnoid hæmorrhage, p. 302). If the blood Wassermann reaction is positive, anti-syphilitic treatment should be given with iodides and mercury (see p. 388). Ligation of the common or internal carotid artery may be considered for right-sided lesions.

### Intracranial Hæmorrhage

**Definition.** Hæmorrhage into the brain or meningeal spaces.

**Anatomy.** Blood is brought to the brain by the two vertebral and the two internal carotid arteries. The vertebral arteries pass along the ventral surface of the medulla, and unite at the lower border of the pons to form the basilar artery. At the upper border of the pons the basilar artery divides into the two posterior cerebral arteries. These are connected with the anterior cerebral arteries by the posterior communicating arteries, and in this way there is formed the circle of Willis, lying in the interpeduncular space on the base of the brain. The internal carotid arteries give off the anterior cerebral arteries, and are then continued as the middle cerebral arteries. The

anterior cerebral arteries are connected by the anterior communicating artery (see Fig 26). The anterior cerebral artery supplies the first frontal convolution, part of the second frontal and the upper part of the ascending frontal and parietal convolutions. The middle cere-

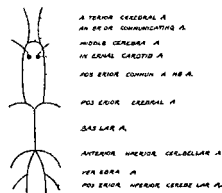


FIG 26. DIAGRAM OF THE CIRCLE OF WILLIS.

bral artery supplies the motor and sensory areas of the cerebral cortex, including the speech area (see p 319). The posterior cerebral artery is distributed to the ventro-mesial portions of the temporal and occipital lobes. Central branches enter the brain substance from the anterior, middle and posterior cerebral arteries, and from the circle of Willis. The lenticulo-striate branch of the middle cerebral artery is distributed to the external capsule, the

lenticular nucleus, the anterior part of the internal capsule and the caudate nucleus. It is a frequent site for a cerebral hæmorrhage. The lenticulo-optic branch supplies the posterior part of the internal capsule. The anatomy of the cranial venous sinuses is given on p 307.

The chief types of intracranial hæmorrhage will now be described.

### Extradural or Epidural Hæmorrhage

**Etiology.** This is associated with concussion and injury to the skull and is also met with in hæmorrhagic pachymeningitis (see p 302).

**Clinical Findings.** At the onset the patient is generally unconscious, and convulsions may occur according to the part of the brain stimulated by the hæmorrhage. He may then recover consciousness for a few hours and appear much better. Recovery may then take place, often associated with a post-concussional state, or this 'lucid interval' be followed by coma and death.

**Treatment.** When the hæmorrhage is due to trauma and there is evidence that the bleeding is continuing, the skull should be trephined and an endeavour made to ligature the bleeding vessel and evacuate the clot.

### Concussion and the Post-Concussional State

**Pathology.** The nature of the concussional cerebral changes due to head injuries remains obscure, despite much experimental investigation. Perivascular petechial hæmorrhages, resulting from diapedesis, may be found usually in the cortex and often in the subjacent white matter and in the grey matter of the brain and brain stem.

**Clinical Findings.** After a minor injury the patient may be temporarily unconscious, exhibiting a flaccid paralysis. The pulse is feeble, temperature subnormal, respirations shallow and slow, and the pupils are dilated. Recovery of consciousness may be rapid or gradual. The

patient may then be unaware of the accident, or he may remember the accident and what happened for a short time after it. He may now pass into an automatic state during which he performs actions of which he has no subsequent recollection. The boxer who fights while "punch-drunk" affords an example of automatism. After a severe injury the patient immediately loses consciousness, and days or even months may elapse before it is fully restored. Certain stages are described in the process of recovery. These may overlap, and all may not be present. The initial coma and flaccid paralysis are followed by restlessness and stupor, which persist for a few days. This may pass into a stage of excitement, bewilderment and delirium in which the patient cannot feed himself, and is incontinent. This stage may last for several weeks or months, and may be interrupted by short periods of stupor. About this time the patient can usually talk more rationally and answer questions. The characteristics of this traumatic psychosis are a disorientation in time and space, retrograde amnesia, speech disturbances, confabulation, and perseveration. Later there is a marked defect of memory for recent events (Korsakow's psychosis). Traumatic hysteria is very liable to follow head injuries. Residues which may persist for some time include headache, difficulty in concentration, abnormal fatigability, forgetfulness and intolerance of even small amounts of alcohol.

### Acute Subdural Hæmatoma

**Etiology.** This results from the rupture of veins running across the subdural space, perhaps from the result of a slight blow on the head.

**Clinical Findings.** The symptoms occur within a few hours of the injury. Sudden attacks of coma with evidence of upper motor neurone lesions, followed by equally sudden return to consciousness and disappearance of physical signs, are very suggestive. Death is likely to occur unless a bilateral subtemporal trephining is performed.

### Chronic Subdural Hæmatoma

**Etiology.** This is as for acute subdural hæmatoma.

**Clinical Findings.** After a latent period of a few weeks or months the patient complains of intermittent headache and mental dulness, gradually increasing in severity. Later, cerebral irritation may ensue, with convulsions or hemiparesis. Papilloedema is not constantly present. The cerebro-spinal fluid may be under increased pressure and contain blood. The combination of signs of increased intracranial pressure, with a normal pressure of cerebro-spinal fluid, is very suggestive of a subdural hæmatoma. The hæmatoma is often situated in the parietal region, and may be bilateral.

**Treatment.** The clot should be removed surgically. It is usually advisable to explore both sides of the brain.

### Spontaneous Subarachnoid Hæmorrhage

**Etiology.** Subarachnoid hæmorrhage is usually due to rupture of a congenital or mycotic cerebral aneurysm.

**Clinical Findings** A congenital intracranial aneurysm usually causes no symptoms until it begins to leak. Slight degrees of bleeding may give rise to headache. In more severe cases the patient experiences intense headache and nausea, vomits and rapidly loses consciousness. *On Examination:* The patient is semi-conscious and looks very ill. The pulse is frequent, breathing stertorous, and the temperature may rise to 101° F or higher. The abdominal reflexes are usually absent, and an extensor plantar response may be present on one or both sides. There may be a certain amount of neck rigidity or head retraction, and convulsive movements of the extremities. The pupils are dilated, and papilloedema, retinal or subhyaloid hæmorrhages may be seen on ophthalmoscopic examination. The urine may contain a considerable amount of albumin for a few days. Glycosuria is present at times, with traces of acetone. If a lumbar puncture is performed, the fluid is found to be uniformly mixed with blood which does not clot on standing. Red cells disappear from the fluid about 7 days after the hæmorrhage has ceased. The bleeding may stop and the patient recover, having various sequelæ, such as headache, disturbance of vision and of mentality, or certain paralyses. In other cases the hæmorrhage continues, generalised bronchitis and œdema of the lungs develop, and the patient dies in the course of a day or so. *The anterior choroidal syndrome* may quickly follow a subarachnoid hæmorrhage. This is characterised by contralateral hemiplegia, hemianæsthesia and hemianopia.

**Prognosis.** Over 60% of cases of subarachnoid hæmorrhage are fatal.

**Treatment.** An initial lumbar puncture is advisable to establish the diagnosis, but it is usually wise not to repeat the lumbar puncture as a routine measure as this may cause the bleeding to recur. It should, however, be repeated if the coma deepens or the blood pressure rises, and if there are convulsions, or headache which is not relieved by morphine. If there is pulmonary œdema, atropin sulph gr 1/100 and morphin sulph gr  $\frac{1}{4}$  should be injected subcutaneously.

### Intracerebral Hæmorrhage

**Definition.** Hæmorrhage into the substance or ventricular system of the brain.

**Etiology.** Hæmorrhage results from rupture of a cerebral artery or aneurysm, or of capillaries, cortical cerebral veins or venous sinuses. Exciting causes include muscular strain and trauma to the skull.

**Predisposing causes** 1 Age. Usually over 40 years. In children hæmorrhage may occur from rupture of cortical veins in whooping-cough, there may be hæmorrhage into a glioma or rupture of an intracranial aneurysm, in infants, hæmorrhage may be due to birth injuries, or rupture of an intracranial aneurysm. 2 Sex. Males predominate. 3 Heredity. Cerebral hæmorrhage tends to run in families. 4 Arteriosclerosis, angiospasm, high blood pressure and chronic nephritis. 5 Blood diseases such as leucæmia or purpura.

**Pathology.** The lenticular branches of the middle cerebral artery are most frequently affected, the lesion being situated commonly in

the thalamus, internal capsule and corpus striatum. Spread to the ventricular system is frequent. In blood diseases, hæmorrhage may occur by diapedesis. In all cases of cerebral hæmorrhage, bleeding usually continues until the patient dies. Congenital aneurysms may, however, leak from time to time before the fatal hæmorrhage occurs. Further an old blood cyst may be found in the brain at autopsy, which indicates the site of a previous hæmorrhage from which the patient has recovered. Meningeal hæmorrhage due to birth injury may lead to atrophy or to the formation of cysts in the brain (porencephaly). The pathology of intracranial aneurysms is considered on p. 298.

**Clinical Findings.** There may be a history of previous slight attacks or "strokes" from which the patient has recovered to a varying degree. These are usually due to cerebral thromboses. The onset is generally sudden, the patient falling unconscious with the apoplectic stroke. Less often there are prodromal symptoms, such as headache, giddiness, vomiting, disturbance of speech, or tingling and weakness in a limb. The results produced vary with the site of the lesion :

1. *Capsular Hæmorrhage.* This is the most common variety, the bleeding starting external to the internal capsule.

*On Examination :* The patient is unconscious, the face is usually flushed, cyanosed and sweating, rarely it is pale. The breathing is stertorous, and the cheek on the paralysed side may be blown in and out with respiration. There is no movement of the limbs, which are flaccid. On lifting up an arm or leg, a greater degree of flaccidity may be detected on the paralysed side. In the early irritative stage there may be conjugate deviation of the eyes, which look away from the site of the lesion in the brain, later with a paralytic lesion the deviation is in the opposite direction towards the site of the lesion. The corneal, superficial and deep reflexes are lost. There may be incontinence or retention of urine, and incontinence of fæces. The pulse is full and bounding, and may be slow or rapid. The temperature is usually subnormal at the onset, and the blood pressure is raised. The urine often contains albumin, and sugar may be present. The cerebro-spinal fluid contains blood if the hæmorrhage has extended into the lateral ventricle. If the patient survives he will pass in a few days into a post-apoplectic stage, described on p. 306 under the title "chronic residual hemiplegia."

2. *Cortical Hæmorrhage.* This is rare. The patient does not generally lose consciousness. Convulsions or paralysis of one or more limbs, aphasia or hemianopia may develop according to the site of the lesion.

3. *Thalamic Hæmorrhage.* This will produce the thalamic syndrome (see p. 312).

4. *Mid-brain Hæmorrhage.* There may be paralysis of the III nerve, with hemiplegia on the opposite side (Weber's syndrome). There may also be anæsthesia of the paralysed side of the body.

5. *Pontine Hæmorrhage.* The patient rapidly becomes comatose. There may be convulsions of the legs, with vomiting, Cheyne-Stokes breathing, and pin-point pupils. Conjugate deviation of the eyes may

also be noted, in a direction opposite to that characteristic of a supranuclear lesion (see p 366) The pupils may dilate before death The temperature rises to 106° F or higher, and the patient dies within a few hours

6 *Medullary Hæmorrhage* The patient usually dies in coma within a few hours before signs of bulbar paralysis are apparent

7 *Ventricular Hæmorrhage* This is usually secondary to capsular hæmorrhage causing rapid death There may be head retraction with rigidity or spasms of the arms or legs

8 *Cerebellar Hæmorrhage* (See p 353)

**Differential Diagnosis** The diagnosis includes a consideration of cerebral thrombosis cerebral embolus a hypertensive cerebral attack (see p 267), and other causes of coma In cerebral thrombosis (see p 300) the onset is more insidious and more likely to occur at night The patient may experience numbness or tingling in the limb, followed during the course of a few hours by paralysis The cerebro spinal fluid does not contain blood In cerebral embolus (see p 307) the onset is usually sudden but often the patient does not lose consciousness unless an extensive area of brain is involved There may be aphasia monoplegia hemiplegia or supranuclear paralysis of the VII cranial nerve The age incidence is often lower than in hæmorrhage and usually a cardiac lesion, such as mitral stenosis or auricular fibrillation is present Cerebral thrombo phlebitis, secondary to systemic thrombosis elsewhere may closely simulate cerebral embolus The spread of infection is considered to be by mural thrombo phlebitis along the intravertebral veins The lungs are not often affected and a mistaken diagnosis of paradoxical embolus (see p 307) may be made Other causes of coma include uræmia, diabetes mellitus cerebro spinal meningitis, opium and alcohol In some cases epilepsy and hysteria will require exclusion In uræmia the urine contains protein and casts, and the percentage of urea in the blood and cerebro spinal fluid is generally raised In diabetic coma the breath smells of acetone, and the urine contains sugar and acetone bodies In cerebral hæmorrhage, although sugar may be present in the urine, acetone bodies are absent In hypoglycæmic coma the patient may have convulsions but a history of diabetes with insulin administration can be obtained and consciousness is usually restored by the subcutaneous injection of 1 mil of liq adrenal hydrochlor Examination of the cerebro-spinal fluid enables a diagnosis of cerebro-spinal fever to be made In opium poisoning the pupils are pin point, and some evidence of the administration of the drug or its derivatives can usually be found Alcohol poisoning may cause difficulty, as the patient may have a hæmorrhage while under the influence of alcohol, and so he should always be kept under observation for 24 hours In epilepsy the unconscious and convulsive phases are generally of comparatively short duration, and if there is resultant hemiplegia (Todd's paralysis) recovery is complete The nature of a hysterical attack is usually clear (see p 317)

**Course and Complications** It is doubtful if a patient can survive a cerebral hæmorrhage, in some cases, after an apparent improvement

with recovery of consciousness, there is recurrence of coma followed by death due to spread of the bleeding into the lateral ventricle.

**Prognosis.** A cerebral hæmorrhage is usually fatal in from 2 to 48 hours.

**Treatment.** The patient should be propped up in bed, all tight clothing loosened, dentures removed and an airway maintained by turning him slightly on one side. If there is marked venous engorgement in the neck, and cyanosis, 10 oz. of blood should be removed from a vein and the bowels opened by an enema. A lumbar puncture should be performed. If blood is present, the diagnosis of hæmorrhage is confirmed; if it is absent, stimulants, such as Coramine (nikethamidum B.P.Add.) 1·5 mil. should be administered subcutaneously six-hourly. If the patient survives, nasal feeding is often required for a time. Catheterisation may be necessary.

### Cerebral Thrombosis

**Definition.** Coagulation of blood in the cerebral vessels.

**Etiology.** *Predisposing causes:* Arteriosclerosis, syphilitic endarteritis, low blood pressure, chronic nephritis, slowing of the circulation in various debilitating conditions, chlorosis, septicæmia, encephalitis lethargica, and polioencephalitis. Cerebral thrombosis may also result from trauma, such as a wound of the brain, or develop in the neighbourhood of a cerebral tumour, or be secondary to a cerebral embolus. Adults over middle age are usually affected, syphilis being the commonest cause in young adults.

**Pathology.** The middle cerebral artery is most frequently affected. The resultant infarct may be red or white. The central arteries are end-arteries, and so softening of the brain with degeneration of nerve elements usually results from their obstruction, and later a scar or cyst may form. The anastomoses are better developed for the cortical arteries.

**Clinical Findings.** If thrombosis affects a large artery the onset of symptoms is acute with coma. When smaller vessels are involved there are often prodromal symptoms. Thus the patient may complain for a few days of headache, giddiness, disturbance of speech, numbness or tingling of a hand or arm, with subsequent paralysis. In some cases there are convulsions at the onset.

**On Examination:** If a large vessel is blocked, the patient is comatose and the clinical findings resemble those described for cerebral hæmorrhage. The signs depend upon the site of the lesion. 1. *Middle cerebral artery thrombosis.* There is usually hemiplegia. At the onset the affected side of the body is flaccid and the superficial and deep reflexes are abolished. The lower half of the face is affected and also the tongue, so that on protrusion it deviates towards the paralysed side. The muscles of mastication, deglutition and the trunk muscles are not usually paralysed. There is some weakness in turning the head and eyes to the paralysed side. In a day or so the plantar response is extensor on the affected side, and the abdominal reflex is absent on the paralysed side, but present on the sound side. 2. *Anterior cerebral artery thrombosis.* There may be no symptoms, or dementia

may ensue. The grasp reflex' (see p 311) may be present in one or both feet. Contralateral crural monoplegia may rapidly develop.

3 *Posterior cerebral artery thrombosis* There may be homonymous hemianopia, the patient having difficulty in avoiding objects on his blind side. Hemianæsthesia may also be present.

4 *Internal carotid thrombosis* This causes coma and rapid death.

5 *Basilar artery thrombosis* This produces symptoms resembling those described for pontine hæmorrhage (see p 303).

6 *Vertebral artery thrombosis* This may produce symptoms of acute bulbar paralysis (see p 394).

**Differential Diagnosis** This is as described for cerebral hæmorrhage (see p 301).

**Course and Complications** After the acute phase of hemiplegia has passed in cases of middle cerebral artery thrombosis the patient enters the stage of chronic residual hemiplegia. Recovery of certain movements is obtained the arm being always more paralysed than the leg. Thus there is power of extension of the hip and knee and of plantar flexion of the ankle and toes. As regards the arm the patient is able to abduct and elevate it and to flex the elbow, wrist and fingers. The muscles producing these movements become spastic and their deep reflexes are increased due to the uncontrolled activity of the extra pyramidal motor paths (see p 280). Ankle clonus and knee clonus are frequently obtained and the plantar response remains extensor. The abdominal reflex may return on the paralysed side. There are also certain associated reactions thus if the patient yawns he may extend his wrist and fingers and raise his hand in front of his face, performing movements involuntarily which he cannot achieve voluntarily.

**Prognosis** This is always unfavourable. Recurrent thrombosis or a fatal cerebral hæmorrhage is liable to ensue.

**Treatment** The patient should be kept in bed at the onset, and stimulants administered such as tne nuc vom m 10 to 15 in an alkaline mixture t.i.d. or subcutaneous injections of Coramine (mket hamidum B.P. Add) 1.5 ml t.i.d. The Wassermann reaction should be determined and if positive a course of anti-syphilitic treatment given (see p 388). Splints should be applied to the affected arm to prevent flexion of the wrist and fingers and the leg should be maintained rotated inwards with the ankle dorsiflexed by means of sandbags. Gentle massage and passive movements should be given in the course of a few days. As soon as possible the patient should be encouraged to perform active movements, but electrical stimulation of the muscles is always contra indicated.

### Cerebral Embolus

**Definition** Obstruction of a cerebral artery by an embolus.

**Etiology** The embolus may be derived from the following sources

- 1 A valve of the heart, especially the mitral and less frequently the aortic valve. This occurs in chronic endocarditis.
- 2 The left auricle or ventricle a portion of clot being detached in mitral stenosis auricular fibrillation or flutter or in diphtheria.
- 3 The endocardium in malignant or subacute infective endocarditis.
- 4 The aorta, in aneurysm or



atheroma. 5. The pulmonary veins, in suppuration of the lungs. 6. The systemic veins, if there is a patent foramen ovale (paradoxical embolism). *Predisposing causes:* 1. Age: Adults and young people. 2. Sex: Females predominate.

**Pathology.** The left middle cerebral artery is most often obstructed, the effects produced resembling those described for cerebral thrombosis (see p. 305).

**Clinical Findings.** The onset is sudden, and there are no prodromal symptoms. There may be convulsions, aphasia, monoplegia, hemiplegia, visual disturbances, or facial palsy of the upper neurone type, according to the part of the brain affected. Consciousness may or may not be lost, depending upon the extent of brain involved. Carotid hemiplegia is due to an embolus obstructing the internal carotid artery, there is homolateral blindness and heterolateral hemiplegia. In many cases an associated cardiac lesion is present, and in some instances the embolus forms during the administration of quinidine (see p. 217).

**Differential Diagnosis.** This is as described for cerebral hæmorrhage.

**Course and Complications.** In carotid hemiplegia there is often restoration of function due to establishment of a collateral circulation through the circle of Willis. Recurrent emboli are liable to form. In the majority of cases the course of the lesion resembles that described for cerebral thrombosis (see p. 306).

**Prognosis.** There is usually recovery of the use of the leg in hemiplegia, but the hand and arm often remain permanently paralysed, and speech may be deranged, especially when the patient is excited. Death results if a large area of the brain is deprived of its blood supply.

**Treatment.** This is as described for cerebral thrombosis (see p. 306).

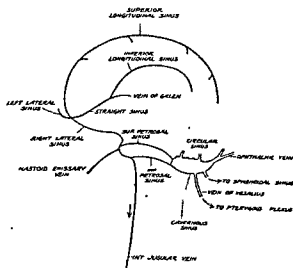


FIG. 27. DIAGRAM SHOWING THE CRANIAL VENOUS SINUSES.

### Sinus Thrombosis

**Definition.** Coagulation of blood in the cranial venous sinuses.

**Etiology.** 1. *Primary sinus thrombosis.* This is an aseptic process. In infants it may be due to marasmus or to congenital syphilis, and in adults to chlorosis and to wasting diseases such as tuberculosis, carcinoma

or enteric fever. The superior longitudinal sinus is usually affected.

**2 Secondary sinus thrombosis** This results from sepsis, as (a) in the middle ear (lateral sinus thrombosis), (b) in the cranial air sinuses, (c) in the skull due to caries or a fracture, (d) in the orbit, nose or upper lip. Thus it may follow a boil, carbuncle or insect bite (cavernous sinus thrombosis). In other cases the infection spreads from the ear, mouth or throat. It may also be a manifestation of thrombo phlebitis migrans or be secondary to peripheral systemic thrombosis which has spread to the brain along the intravertebral veins (see p. 304).

**Anatomy and Pathology** The anatomy of the important cranial venous sinuses is indicated in the diagram (see Fig. 27). The walls of the sinuses consist of dura mater. The blood enters the sinuses from the brain and the meninges. The direction of flow in the emissary veins is uncertain. The blood passes from all the sinuses into the internal jugular veins. There are five single sinuses, namely, the superior longitudinal, the inferior longitudinal, the straight, the circular, and the basilar sinuses. There are six paired sinuses, namely, the lateral, the occipital, the cavernous, the superior petrosal, the inferior petrosal, and the sphenoparietal sinuses.

From the medical point of view the most important are the cavernous sinus, the lateral sinus and the superior longitudinal sinus. The cavernous sinus is in close proximity to the sphenoidal air sinus, and only separated from it by a thin layer of bone. The internal carotid artery and the VI nerve are on its inner side, and the III, IV and V nerves pass

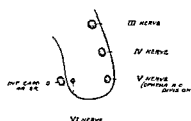


FIG. 28. DIAGRAM SHOWING THE CAVERNOUS SINUS AND ITS NERVES

through its outer wall (see Fig. 28). The ophthalmic veins enter it in front and it is also in communication anteriorly with the frontal and supra-orbital veins. The superior and inferior petrosal sinuses connect it with the lateral sinus behind. The circular sinus connects the two cavernous sinuses. The superior longitudinal sinus receives blood from the brain, and obstruction is liable to cause gross cerebral disturbance. Infection probably spreads by means of infected blood clot or along the sheath of the I and VIII nerves. There is no lymphatic path. At autopsy a clot may be found adhering to the wall of the sinus and in some cases it extends into the jugular and subclavian veins, or even into the superior vena cava. Organisms, such as streptococci or pneumococci, may be present in the clot.

**Clinical Findings** *Cavernous Sinus Thrombosis* In a typical case the patient has noticed a sore place or small boil on the upper lip or the inside of the nose. It may have been opened surgically. A day or so later the patient complains of frontal headache with malaise and drowsiness. Later, there may be pain in the side of the face or eye, and the vision may fail.

**On Examination** At the onset the temperature is found to be raised to about  $101^{\circ}\text{F}$ , and the pulse to 90 or 100. Definite signs soon appear

and the patient becomes very ill. There is protrusion usually of both eyes, swelling of the eyelids and œdema of the conjunctivæ, which causes them to protrude between the closing eyelids (chemosis). The eyelids themselves are œdematous, and may be bluish. The swelling may spread to the forehead and side of the face. External oculomotor paralysis is commonly found, the VI nerve being often first affected, and the pupils may be dilated and fixed. The infection sometimes travels back to the lateral sinus, causing pain behind the ear and some œdema over the mastoid process. It may spread even further to the internal jugular vein, when there is stiffness, pain and swelling of the neck. The cerebro-spinal fluid: This is often under increased pressure, and contains an excess of polymorphonuclear cells, and organisms.

*Lateral Sinus Thrombosis.* The patient frequently has been suffering from chronic suppurative otitis media or a mastoid operation may have been performed. He complains of headache or vomiting.

*On Examination:* There may be no otorrhœa, the temperature rises, and is swinging in type, but the pulse is often slow. Frequently there are no other signs, but papilloœdema may be present. If the infection spreads to the internal jugular vein, the patient will notice difficulty in turning the head to the opposite side, and the vein is often felt thrombosed. There may also be œdema over the mastoid process.

*Superior Longitudinal Sinus Thrombosis.* The patient complains of headache, vomiting or bleeding from the nose.

*On Examination:* The anterior fontanelle is seen bulging in infants, and there is often enlargement of the veins on the front and side of the head. Convulsions and spastic paralysis of both legs may rapidly ensue. The patient usually becomes delirious and incontinent. The cerebro-spinal fluid: In infants this is usually clear and under increased pressure with an increase of lymphocytes.

*Differential Diagnosis.* In cavernous sinus thrombosis the bilateral proptosis with swelling of the eyelids is very characteristic. Proptosis associated with retro-orbital tumours is usually afebrile. It should always be remembered that with sphenoidal sinusitis, in which the cavernous sinus is not affected, there may be swelling of the eyelids, bulging eyes and headache. Transillumination, X-ray examination and exploratory puncture should serve to differentiate. Further, cavernous sinus thrombosis may occur without proptosis. It is not usually possible to diagnose lateral sinus thrombosis with certainty apart from exploratory operation. Examination of the cerebro-spinal fluid will indicate whether meningitis is present, but meningitis may also occur as a complication of the sinus thrombosis.

*Course and Complications.* The course is usually rapid, complications include meningitis, cerebral abscess and pyæmia.

*Prognosis.* This is very grave, the patient frequently dying in a few days.

*Treatment. Prophylactic.* Septic spots on the upper lip and nose should be fomented and not squeezed, and early surgical intervention is dangerous. When pus definitely forms it should be evacuated through a small incision. All cases of otorrhœa should be adequately treated.

*Curative.* The only chance of a cure is by operation, the sinus being exposed and the clot removed. There is no treatment likely to prove successful for primary cases.

### Intracranial Tumours

**Definition.** New growths, granulomata and cysts arising in the brain, meninges or interior of the skull.

**Etiology.** In some cases of intracranial tumour there is a history of a blow on the head, followed by the signs of the tumour. These signs may result from the occurrence of œdema or hæmorrhage in or around a pre-existent tumour. *Predisposing causes.* 1 Age. The majority of tumours occur in young or middle-aged people. Glioma and tuberculoma may affect children. 2 Sex. Males predominate slightly.

**Pathology.** The following tumours may occur, and they are grouped approximately in their order of frequency. Glioma (including astrocytoma, multiple spongioblastoma, medulloblastoma, ependymoma, etc), pituitary adenoma, meningioma (endothelioma), acoustic neuro-fibroma, congenital cysts, secondary malignant growths such as sarcoma, carcinoma and hypernephroma, gumma, tuberculoma, vascular tumours such as telangiectasis, venous angioma, arterio-venous aneurysm and angio-blastoma, papilloma, cholesteatoma, psammoma, fibroma, chloroma and lipoma. Primary sarcoma and actinomycosis are rare. In children a tuberculoma is the commonest tumour. Cysts include a dermoid, hydatid, cysticercus cellulosæ, serous cyst, blood cyst, colloid cyst of the third ventricle and congenital cysts. The glioma is ectodermal in origin, it tends to infiltrate the cerebral substance, is vascular and liable to hæmorrhage or to cystic degeneration. The meningioma arises from arachnoid connective tissue. It invades bone with secondary bony overgrowth, and compresses the brain. Carcinoma of the brain is usually secondary to a growth in the breast, lung or kidney. The increase in size of the tumour causes cerebral anæmia, and the anoxia thus produced may result in localised epileptiform fits. Increased intracranial tension and hydrocephalus are obstructive phenomena. The frontal region is the site of about 20% of brain tumours, and about 17% occur in the cerebellum.

**Clinical Findings.** The initial symptoms are usually insidious, but at times an intracranial emergency provokes an acute onset. Thus a child may be apparently perfectly well, when suddenly he falls unconscious owing to a hæmorrhage into a glioma. The symptoms and signs are divided into two groups, general and focal. Often the general symptoms are first noted, but in some cases, especially in cerebello-pontine tumours and in the aged, general symptoms are inconspicuous. The general symptoms and signs are an index of raised intracranial pressure, and include headache, vomiting and papilloedema. These three are present together in about 60% of all cases of cerebral tumours, and headache alone in about 80% of all cases. The headache. At first this is paroxysmal and often worse on stooping or at night. Later it becomes persistent. The site of the pain rarely serves to localise the

tumour. The skull may be tender on percussion over the tumour. The headache is due to stimulation of the dural branches of the V nerve, by stretching of the dura. The vomiting: This is typically projectile, occurring apart from meals, at night or early in the morning. At times it is not projectile and there is also nausea. Vomiting results from stimulation of the medullary centre. Papilloedema: 'This may' exist in a marked degree without visual disturbance, later, sight is blurred and blindness ensues with atrophy of the optic nerves. The papilloedema is due to the pressure of the cerebro-spinal fluid in the sheath of the optic nerve on the central vein of the retina. The pulse tends to be slow, the temperature subnormal, and respiration of the Cheyne-Stokes type (see p. 226). With tumours of the posterior fossa there is nearly always tachycardia. In some cases, if the skull is shaved, dilated veins are apparent in the scalp over the tumour, and in children the sutures may be widened. A radiogram of the skull may show thinning of the bone (beaten silver appearance), calcification of a meningioma, or displacement of the pineal body. Ventriculography or encephalography (see p. 318) may reveal an obstruction in, or displacement of the ventricular system of the brain. Arterial encephalography (angiography) will show blood vessels displaced by a tumour. The electro-encephalogram (see p. 288) may also aid in the localisation of cerebral tumours. Slow delta waves arise in the cerebral tissue surrounding the tumour, when the tumour causes progressive destruction of cortical tissue. Discrete and non-progressive cortical lesions, or deep tumours not affecting the cerebral hemisphere or the cortex, are not likely to be unmasked by electro-encephalography. The site of origin of the abnormal delta waves can be localised by a series of electro-encephalograms. In all cases of cerebral tumour it is dangerous to perform lumbar puncture if the intracranial pressure is raised, as there is risk of death from incarceration of the cerebellum and medulla in the foramen magnum. A tumour distant from the pituitary fossa may cause signs of hypopituitarism. The increased pressure of the cerebro-spinal fluid distends the floor of the third ventricle and causes pressure on the pituitary. *Focal symptoms and signs* vary according to the site of the tumour and will be considered regionally.

1. *Pre-frontal Region.* An alteration in mentality may be noted. There may be drowsiness, lack of orientation for time, or a tendency to make jokes (*witzelsucht*). At times such signs as yawning, stiffness of the neck muscles, tremor of the homolateral hand, diminution of the heterolateral abdominal reflex, frequent nose rubbing and incontinence of urine or faeces at night may be found. The "grasp-reflex" ("forced grasping" or "groping") may be demonstrable. An object placed on the palm of the hand, on the opposite side to the cerebral lesion, between the thumb and first finger, causes a grasping movement. A similar "grasp-reflex" in the foot may sometimes be elicited with a tumour situated in the medial aspect of the frontal lobe and in the corpus callosum. The "grasp-reflex" of the foot is elicited by light pressure applied to the plantar surface of the foot, especially in the area of the ball of the big toe. The reflex consists in flexion and adduction of the

toes, of a tonic nature, lasting for 15 seconds or longer. There may also be inversion of the foot. The reflex is normally present in all infants under the age of 9 months, and disappears in the vast majority of cases by the age of 2 years. It is present abnormally when a lesion interferes with the fronto pontine fibres. It is therefore probably a release phenomenon, and not a reflex, and consists of low grade volitional movements. The movements do not occur in infants during sleep, nor in adult patients when unconscious. Pressure symptoms include anosmia, resulting from pressure on the olfactory nerve or bulb, and central scotoma with primary optic atrophy may occur on the side of the lesion and papilloedema in the other eye.

2 *Ascending Frontal Region* Irritative or paralytic phenomena may be noted. A cortical tumour may cause Jacksonian epilepsy, with movements starting at the angle of the mouth, or in the thumb or big toe on the opposite side. There may be conjugate deviation of the eyes away from the affected side. With subcortical tumours there may be paralysis of an arm, leg or part of the face. With left sided lesions aphasia may be present.

3 *Parietal Region* The patient may notice an awkwardness in movement, or blunting of sensation on the opposite side of the body. Pain and temperature sensation are not affected, but there may be loss of postural sense in the opposite limbs, with astereognosis and disability in the discrimination of two points, as judged by the compass test. Word blindness may also result from a tumour involving the supra-marginal or angular gyri on the left side.

4 *Temporo sphenoidal Region* If the uncinate gyrus is involved there may be uncinate fits, the patient passing into a dreamy state with unpleasant sensations of taste or smell, and at times movements of the lips. The growth may extend into the optic radiation, with resultant quadrant homonymous hemianopia (see p 360). When the first left temporal convolution is affected the patient may experience difficulty in naming objects, although he recognises the correct name when it is said (nominal aphasia).

5 *The Occipital Region* Involvement of the area around the calcarine fissure may result in homonymous hemianopia. Visual hallucinations, such as flashes of light may also occur.

6 *The Internal Capsule* Involvement of the anterior limb causes hemiplegia on the opposite side. If the tumour affects the posterior limb there will be contralateral hemianesthesia or hemianopia.

7 *The Optic Thalamus* A tumour destroying the thalamus may result in the "thalamic syndrome". There is weakness of the opposite side of the body and choreic or athetoid movements may be seen. Severe pains may be felt on the opposite side of the body and sensation is altered. Such stimuli as tickling or scraping or the extremes of heat and cold may provoke very severe reactions although the patient is not able to differentiate between the blunt and sharp end of a pin. There is also astereognosis and loss of postural sense on the opposite side.

8 *The Third Ventricle* A colloid cyst produces intermittent

symptoms, especially very severe headache which may terminate in unconsciousness. There is papillœdema with gradual failure of vision.

9. *The Mid-brain.* A tumour in the region of the anterior corpora quadrigemina may cause bilateral ptosis, with weakness of up and down movements of the eyes and a sluggish pupil reaction. There may also be hemiplegia on the opposite side, and bilateral ataxy of the arms, if the decussating superior cerebellar peduncles are involved. Bilateral deafness may result from involvement of the posterior corpora quadrigemina.

10. *The Pons.* The V, VI or VII nuclei may be affected with paralysis of the muscles supplied by them (see pp. 362, 367, 371). There may also be hemiplegia and possibly hemianæsthesia on the opposite side of the body. The pupils are often small, due to interference with the impulses which cause dilatation. These pass down the mid-brain and cord to emerge in the cervical sympathetic.

11. *The Medulla.* There may be unilateral or bilateral paralysis of the IX, X, XI and XII nuclei, with disturbance of swallowing, mastication and speech. Both pyramidal tracts may also be involved, with bilateral hemiplegia.

12. *The Interventricular Region.* It is not usually possible to diagnose during life tumours arising here.

13. *Cerebellar and Cerebello-pontine Regions.* Tumours of these areas are considered on p. 355.

14. *The Pituitary Body.* These tumours are considered on p. 669.

*Differential Diagnosis.* The diagnosis of a cerebral tumour is often very difficult. Thus localised or generalised convulsions may occur for years with a subcortical tumour. These are usually considered to be due to epilepsy or to hysteria, until papillœdema appears. Vomiting may also be considered a hysterical manifestation. Papillœdema may be met with in nephritis, septicæmia or severe anæmia. When there are localising signs in the brain other conditions such as a vascular lesion or an abscess must be considered. An abscess may run an afebrile course, but the blood usually shows a leucocytosis. In acute cases, the acute variety of disseminated sclerosis, encephalitis lethargica, hypertensive encephalopathy, lead encephalopathy, hydrocephalus or abscess secondary to bronchiectasis may all require to be eliminated. The Wassermann reaction is helpful in establishing a diagnosis of a gumma, especially if the response to anti-syphilitic treatment is good. Secondary malignant deposits in the brain are suggested by the discovery of a primary focus elsewhere.

*Course and Complications.* The course is usually steadily progressive, but sudden exacerbations of symptoms due to œdema may be expected. In some cases improvement occurs, owing to absorption of œdematous fluid. Complications include internal hydrocephalus, hæmorrhage, meningitis and secondary hypopituitarism.

*Prognosis.* This is always very grave, and the outlook if surgery is adopted is usually gloomy. Cerebral tumours as a class do not lend themselves to excision owing to their ill-defined margins. The most favourable, as judged by the survival period after operation, is the

pituitary adenoma, for over 70% of patients survive the operation by 7 years. Next come the cerebellar astrocytoma, the acoustic tumour and the meningioma. An operation which removes the tumour, but results in the patient becoming hemiplegic and aphasic can neither be considered successful nor justifiable. The outlook is hopeless with secondary malignant tumours. A gumma often responds to treatment and a tuberculoma may at times become obsolete.

**Treatment** The Wassermann reaction should be determined, and, if positive, a course of treatment given, as described on p 388. If the tumour is localised, accessible and of suitable type, an attempt may be made to remove it surgically. Decompression alone is a palliative measure to save sight and relieve headache. In any case in which an operation is considered inadvisable, the effect of a course of iodides should be tried (see p 168). Headache and vomiting may be temporarily relieved by the rectal injection of 8 oz of 25% mag sulph solution, or by the intravenous injection of 25 mls of 30% sod chlorid solution, or of 50 mls of 50% dextrose solution. These intravenous injections should be given at the rate of 3 mls a minute. Pain is relieved by aspirin gr 10 t.i.d.s. or by morphine injections as required. Vascular tumours, such as a venous angioma and arterio venous aneurysm, should be treated by deep X rays.

### Abscess of the Brain

**Definition** Localised suppuration in the brain.

**Etiology** The abscess may be due to—  
 1 Local trauma. The brain may be injured by a gun shot wound or a stab. In some cases an abscess follows a blow on the head, although neither skull nor scalp are severely damaged.  
 2 Direct spread of infection. The septic focus is most often in the ear, as in chronic otitis media, or in the mastoid cells. In other cases the abscess is secondary to infection of the frontal, ethmoidal or sphenoidal sinuses. Syphilitic or tuberculous caries of the skull, and erysipelas or a carbuncle on the scalp, face or neck, may lead to cerebral abscess.  
 3 Blood borne infection. The septic focus is frequently intra thoracic, such as bronchiectasis, lung abscess or empyema. In other cases the cerebral abscess is secondary to osteomyelitis, puerperal sepsis, infective endocarditis, pyonephrosis, etc.  
 4 Local lesions. A cerebral neoplasm or tuberculoma rarely suppurates. A cerebral abscess is most often met with between the ages of 11 and 35 years.

**Pathology** A solitary abscess is usually due to trauma or to intra thoracic suppuration, and multiple abscesses to extra thoracic suppuration. A localised intracranial abscess is also found either extradurally between the dura and the skull, or subdurally between the dura and pia. The cerebral abscess may be acute or chronic. The acute abscess contains creamy or blood stained material, the chronic abscess is filled with greenish offensive pus and usually has a capsule formed by inflammatory tissue. Various organisms, such as staphylococci, pneumococci, streptococci and the *Bacterium commune* (*B. coli*) may be



present, or the contents may be sterile. The *Actinomyces bovis* (*Streptothrix actinomyces*) is rarely found. The abscess varies in size from a minute spot to that of a large orange. It usually forms in the white matter below the cortex, which is comparatively avascular, between the brain tissue supplied by the end branches of the cortical and central cerebral arteries. When the cerebral abscess is secondary to infection in the soft tissues outside the skull, the infection may spread along thrombosed emissary veins. An abscess of the brain secondary to osteitis of the skull is usually due to spread of infection along the perivascular spaces.

**Clinical Findings.** There is often a history that the patient has suffered from chronic suppurative otitis media for some years. The onset of abscess formation may be heralded by cessation of discharge from the ear. In some cases certain stages can be recognised clinically. These are: 1. *The stage of onset.* This may be of short duration, only 1 or 2 days, during which the patient does not feel well; there may be shivering or even a rigor, with headache and perhaps nausea or vomiting. The temperature rises to 101° F. or higher, but the pulse remains comparatively slow. 2. *The latent stage.* This may last for a few weeks. The patient feels drowsy, and complains of a dull headache, and his appetite is poor.

**On Examination:** Local tenderness may be found on tapping the skull at some point. The pupil may be dilated on the affected side. Mental changes such as delusions, hallucinations, irritability, etc., have been described in some cases. The temperature is usually low, but if it is charted every 2 hours, an irregular swing may be detected just above or below the normal line. The pulse may drop to about 60 for short periods during the 24 hours. The blood: There is usually a leucocytosis of 15,000 or 20,000 per c.mm. The cerebro-spinal fluid: This is often under increased pressure, and there is an increase of polymorphonuclear cells. The chloride figure may be low. The fluid is sterile. 3. *The stage of localised signs.* Whether or not localising signs appear depends upon the site of the abscess. If it is in the temporo-sphenoidal lobe, there may be deafness on the opposite side, or if it is on the left side of the brain there may be word deafness, the patient not appreciating the meaning of spoken words, or having difficulty in naming objects. Pressure on the pyramidal tract in the pons may result in weakness on the opposite side of the body with an extensor plantar response, or loss of the abdominal reflex on the other side of the body. Extension forwards may cause weakness of the lower part of the face on the opposite side. In some cases there is pressure on the III or VI cranial nerves on the same side. An abscess in the occipital lobe may cause homonymous hemianopia, whereas if it is situated in the parietal lobe the patient may lose the power of recognising the shape of objects by touch (astereognosis). An abscess in the pre-rolandic area will cause motor paralysis of the opposite side of the body. The symptoms of a cerebellar abscess are described on p. 350. Some degree of papilloedema may develop. 4. *The terminal stage.* This usually implies the phase of coma. The abscess may rupture intraventricularly, with convulsions, delirium, a high

temperature and rapid action of the heart, followed by death in a few hours. Meningeal rupture is indicated by signs of meningitis.

**Differential Diagnosis** It is often extremely difficult to diagnose with certainty the presence of a cerebral abscess in cases of chronic suppuration in the ear. Mastoiditis and meningitis must be excluded, and they may co-exist with cerebral abscess. The examination of the cerebro-spinal fluid establishes or excludes the presence of meningitis. Further, sinus thrombosis (see p. 309) may cause difficulties in diagnosis. There is usually a sudden onset to the symptoms in sinus thrombosis, with rigors, a high temperature, rapid pulse, and at times local tenderness. With a cerebral tumour (see p. 313), papilloedema is likely to occur early and to be more intense than is the case with a brain abscess. *Leucocytosis is generally absent, and no primary cause for an abscess is discovered.* A hæmorrhage into a glioma may cause acute symptoms, resembling those produced by a brain abscess.

**Course and Complications.** Acute abscesses are usually rapidly fatal. A chronic abscess may be encapsuled for years and give rise to no symptoms until it suddenly bursts into a ventricle or into the subarachnoid space. Complications include pyocephalus, hydrocephalus, cerebral sinus thrombosis, meningitis and septicæmia.

**Prognosis** A cerebral abscess which causes symptoms will result in death unless drained surgically. In any case the prognosis depends upon the primary condition. Abscesses secondary to ear infection do well if diagnosed and treated early.

**Treatment** This is surgical. The abscess must be explored and drained.

### Hydrocephalus

**Definition** Distension of the ventricles of the brain with cerebro-spinal fluid.

**Etiology** Theoretically the distension might arise from various causes such as—  
 1 Over production of cerebro-spinal fluid.  
 2 Blockage of an exit from a ventricle.  
 3 Obstruction to the absorption of cerebro-spinal fluid.  
 The majority of cases in practice are due to 2 or 3. *Predisposing causes*—  
 1 Age. Usually infants or young children.  
 2 Heredity. Congenital hydrocephalus may run in families.

In order to understand how these causes operate it is necessary to outline the physiology of the circulation of the cerebro-spinal fluid.

**Physiology and Pathology** The cerebro-spinal fluid is derived, probably by filtration, from the blood circulating through the choroid plexuses in the lateral, third and fourth ventricles. These vessels lie in invaginated folds of the pia mater. The fluid leaves the fourth ventricle by the foramina of Majendie and Luschka, which are situated in the roof and lateral recesses of the ventricle and passes out into the subarachnoid space, which here constitutes the cisterna magna. It then circulates around the brain and cord. In order to reach the brain it passes through a channel, the chiasma tentorii, in the subarachnoid space, at the level of the tentorium cerebelli and so reaches the cisterna

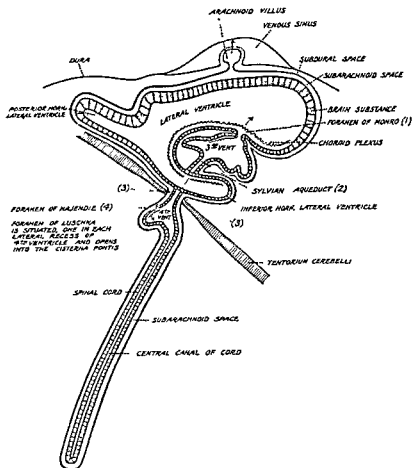


FIG. 29 DIAGRAM INDICATING THE STRUCTURES CONCERNED IN THE PRODUCTION OF HYDROCEPHALUS.

*Obstruction at (1), foramen of Monro, causes distention of one lateral ventricle.*

*Obstruction at (2), Sylvian aqueduct, causes distention of both lateral ventricles and of the 3rd ventricle.*

*Obstruction at (3), tentorium cerebelli, causes communicating hydrocephalus, with distention of the lateral, 3rd and 4th ventricles, a communication being patent through the foramina of Majendie and Luschka, with the subarachnoid space around the cord and the ventricular system. The fluid passing from the 4th ventricle to the subarachnoid space cannot reach the subarachnoid space above the tentorium where absorption normally chiefly occurs*

*Obstruction at (4), foramina of Majendie and Luschka, causes non-communicating distention of the lateral, 3rd and 4th ventricles*

basalis, and passing thence over the brain, absorption occurs, again probably by filtration, through the arachnoid villi into the venous sinuses of the skull. Hydrocephalus may be congenital or acquired, and certain varieties are described.

*External Hydrocephalus.* This is a misnomer; serous fluid accumulates in the subdural space, leading to atrophy of the brain, as in senile atrophy or general paralysis of the insane. The cerebral ventricles may also be dilated, constituting compensatory hydrocephalus.

*Internal or Hypertensive Hydrocephalus* (see Fig. 20). 1. *Increased production of fluid.* It is possible, as mentioned above, that obstruction to the venous circulation in the brain would increase the output of fluid from the choroid plexuses to the ventricles. Thus the great vein of Galen, which drains the choroid plexuses of the lateral and third ventricles, might be compressed by a subtentorial tumour. 2. *Obstructed circulation of fluid.* Obstruction of one foramen of Monro will cause dilatation of the corresponding lateral ventricle. This may be due to a tumour of the choroid plexus, and it may be intermittent. Obstruction of the Sylvian aqueduct, as by a tumour, will result in dilatation of the third ventricle and both lateral ventricles. Obstruction of the subarachnoid space, as by meningeal adhesions, at the level of the tentorium cerebelli, causes a "communicating hydrocephalus." The fluid which leaves the fourth ventricle through the foramina of Majendie and Luschka, cannot pass above the tentorium to be absorbed. Normally only one-fifth of the absorption takes place from the subarachnoid space below the level of the tentorium, and four-fifths above this plane. The pressure therefore rises both in the ventricles, which dilate, and in the subarachnoid space around the cord, as shown by lumbar puncture. Obstruction of the foramina of Majendie and Luschka, as by meningeal adhesions, causes dilatation of the fourth, third and the lateral ventricles. 3. *Defective absorption.* This is not often a cause of hydrocephalus. It may result from an inflammatory obstruction of the arachnoid villi, by a generalised increased intravenous pressure in the skull due to a tumour, or by thrombosis of the superior longitudinal sinus (see otitic hydrocephalus, p. 298).

In congenital hydrocephalus a structural defect may be present, such as a Sylvian aqueduct with an opening in its roof; or a scar may cause obliteration of the aqueduct; or the subarachnoid space may be obliterated by adhesions resulting from a hæmorrhage at birth. Less often the foramina of Majendie and Luschka are obliterated by adhesions. In many cases no cause is found. In acquired hydrocephalus the lesion may result from syphilitic or meningococcal meningitis, cerebral tumours, especially those situated in the posterior cerebral fossa, and rarely from thrombosis affecting the great vein of Galen or the cerebral venous sinuses. The normal amount of cerebro-spinal fluid present is about 150 c.c. (5 oz.), with hydrocephalus this is usually increased 3 or 4 times, but much larger quantities have been found. The lateral ventricles may be greatly dilated, the brain substance being reduced to a thin layer.

*Clinical Findings. Congenital Hydrocephalus.* The head may be

abnormally large at birth causing difficulty in labour, or the enlargement may not be noted until a few days after birth. The skull bones become separated, the scalp is thin and translucent, the veins being prominent and the hair scanty. The infant's face is dwarfed by the protruding forehead, and the lower lids may partly cover the pupils of the eyes, owing to downward pressure on the eyes by the orbital plates of the skull. In a severe case the infant cannot lift its head off the pillow. Other congenital defects may be present, such as hare lip, cleft palate, spina bifida and imperforate anus, etc. The infant may suffer from convulsions and some degree of spastic paraplegia. If the infant survives, mental deficiency or blindness due to optic atrophy may be found.

*Acquired Hydrocephalus* The patient may be a young child or an adult of any age. If the hydrocephalus develops after the skull bones have firmly united there is no enlargement of the head, if bony union is not complete some enlargement may occur. The patient complains of very severe headache, vomiting, disturbance of vision and unsteadiness or giddiness.

*On Examination* Papilloedema may be present, and in some cases there is weakness of the arms or legs of a spastic type with exaggeration of the deep reflexes, and an extensor plantar response.

*Differential Diagnosis* The diagnosis of congenital hydrocephalus usually presents no difficulty, slight cases should not be mistaken for rickets. In cases of acquired hydrocephalus in which there is no enlargement of the skull, the condition closely resembles that of cerebral tumour, which may co-exist. Special tests may be employed to demonstrate the presence and the variety of hydrocephalus. These include

1. *Ventriculography and encephalography* Air is injected into the lateral ventricle, either through the anterior fontanelle, if unclosed, or by trephining the skull. Radiograms will show the air in the ventricles and indicate whether there is free communication between them. Encephalography is performed by lumbar puncture, removing about 6 to 10 c.c. of fluid and then injecting with the syringe a smaller volume of air. This is repeated until about 50 to 100 c.c. of air have been injected. The air passes up the subarachnoid space and infiltrates not only the subarachnoid space over the cerebral cortex, but also passes into the ventricles. A block at any point may thus be demonstrated.

2. *Pressure tests* If a manometer is inserted into the lateral ventricle, and another into the spinal theca, the pressure should be the same if no obstruction is present. In a communicating hydrocephalus the cerebrospinal fluid is often under increased pressure, corresponding with the increased intraventricular pressure. In non communicating hydrocephalus the intraventricular pressure is raised, where the spinal pressure is normal.

3. *Dye tests* If 1 ml. of indigo-carmin solution is injected into the lateral ventricle and a lumbar puncture is performed a quarter of an hour later, in a normal patient the dye is then found in the spinal fluid. In obstructive non-communicating hydrocephalus the dye does not pass to the spinal fluid.

*Course and Complications* Congenital hydrocephalus may pursue

a rapid course or be arrested at any stage. In some cases the children grow up and are remarkably intelligent. Acquired hydrocephalus is usually progressive, but the course must vary with the cause.

**Prognosis.** In congenital hydrocephalus death may occur in a few weeks or months; in other cases arrest takes place. In acquired hydrocephalus arrest may occur, or the disease cause death in a few weeks, months or years. The prognosis depends upon the cause, cases due to tumours are usually hopeless.

**Treatment.** Surgical treatment is still experimental, but suboccipital decompression may be recommended for failing vision in cases of the communicating type. Medical treatment is usually of little avail; deep X-ray radiation may be applied to the skull and diuretic drugs used, such as theophyll. et sod. acetat gr. 3 t.d.s. In some cases small doses of thyroideum, such as gr.  $\frac{1}{4}$  to gr.  $\frac{1}{2}$  t.d.s., appear helpful. If the condition is syphilitic, as suggested by a positive Wassermann test, ung. hydrarg. gr. 60 should be rubbed into the scalp daily, and pot. iod. gr. 5 to 30 given t.d.s. according to the age of the patient.

### Aphasia

**Definition.** Disturbances of speech, of writing, and of the understanding of spoken and written words, due to cerebral lesions.

**Etiology.** The lesion is on the left side of the brain in right-handed

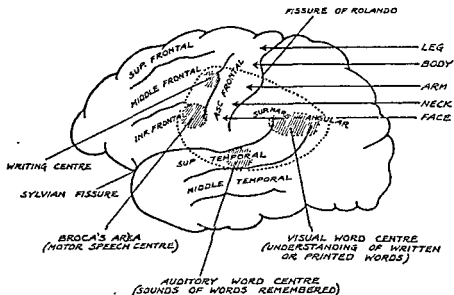


FIG. 30. DIAGRAM OF THE SPEECH CENTRES AND CORTICAL MOTOR CENTRES OF THE BRAIN.

people, and is usually a thrombosis, embolus, tumour, or abscess.

**Physiology and Pathology.** Nothing is known very definitely regarding the physiology of speech. The older physiologists believed in definite cortical centres, such as a motor speech centre, connected with the area of the brain controlling the speech muscles, a motor writing centre connected with the hand, a sensory visual centre and a

sensory auditory centre connected with the eye and ear respectively, the whole area comprising the speech zone (see Fig 30) The modern tendency is to regard the subcortical zone with its association fibres as being intimately concerned with speech and writing faculties Thus Broca in 1861 taught that aphasia is due to a lesion at the posterior end of the left inferior frontal convolution Wernicke (1874) postulated, in addition to the motor centre in Broca's area, a visual sensory centre in the left supramarginal and angular gyri and their subcortical zones, and an auditory sensory centre in the left superior and middle temporal convolutions and the subcortical area Marie (1906) considered that true aphasia is due to a lesion in Wernicke's sensory zone, and that motor aphasia results from a combination of anarthria (disordered articulation) with sensory aphasia Anarthria is due to a lesion in the neighbourhood of the lenticular nucleus and the genu of the internal capsule Thus in some cases of motor aphasia no lesion is found post-mortem in Broca's area, and in motor aphasia there is always some intellectual weakness Head (1910 and subsequently), after extensive study of the subject, introduced certain terms for types of aphasia which are probably cortical in origin These include verbal aphasia with difficulty in word formation, nominal aphasia with incorrect use of nouns in speech or writing, syntactical aphasia with incoherent or jargon speech, and semantic aphasia in which there is a lack of appreciation of the deeper significance of words and phrases The speech area of the brain is supplied by the left Sylvian artery, a branch of the middle cerebral artery The usual lesion is a thrombosis, embolus less often causes aphasia, a hæmorrhage so frequently results in death that aphasia cannot be observed Aphasia of insidious onset may be due to a tumour Transitory aphasia may result from epilepsy, migraine, uræmia general paralysis of the insane or encephalitis lethargica

**Clinical Findings** In the majority of cases the aphasia has a sudden onset and is associated with other symptoms of a "stroke" There are various clinical types of aphasia, such as 1 *Motor aphasia* The patient is unable to speak intelligibly, but the actual muscles involved in speech are not necessarily paralysed Often he can say a few simple words, and he can understand what is said to him 2 *Nominal aphasia* The patient may not be able to name an object which he recognises He can realise, however, whether or not it is correctly named by someone else 3 *Agraphia* The patient cannot write, although the hand muscles which are controlled by the adjacent cortical area in the ascending frontal convolution are not paralysed 4 *Anarthria* Disordered speech may be due to a bilateral cortical lesion, or a bilateral lesion of the internal capsule (pseudo-bulbar paralysis) or to a bulbar nuclear lesion (see p. 304) 5 *Word deafness* Spoken language is not understood, it seems to be a foreign tongue The patient's speech may be a jargon 6 *Word blindness (alexia)* The patient can see written words, but does not appreciate their meaning 7 *Mixed aphasia* In the majority of cases the aphasia is mixed There is a disturbance of general intelligence, as well as of speech, writing and understanding of spoken words, and often the patient is also suffering from hemiplegia

*Apraxia and agnosia* are conditions closely allied to aphasia. In the former the patient is unable to carry out certain complicated movements, such as buttoning up his clothes. In the latter he confuses the use of objects.

*On Examination:* A certain routine should be observed in investigating cases of aphasia. A complete neurological examination must be carried out as detailed above (see p. 283) and any lesion which is detected can often thus be localised. An enquiry should be made as to whether the patient is right or left-handed. The power of speech should next be tested by asking the patient to say how he feels, etc. If he cannot speak intelligibly his power of understanding spoken words should be tested by asking him questions, making him indicate his answers by signs or writing. The power of understanding written or printed words is then determined by giving him a paper to read, and asking him if he understands it. He is asked to write, to repeat words spoken to him, to copy words, pick out named objects, to name objects, and to write from dictation. In this way the various connecting links of the speech area of the brain are tested.

*Prognosis.* This naturally depends upon the nature of the lesion. If it is not a hæmorrhage or a tumour, there is usually a tendency to improvement, but subsequent attacks are liable to occur.

*Treatment.* When the patient has recovered from the shock and the immediate results of the lesion which has caused the aphasia, his power of speech may be slowly improved by the help of a specially trained speech instructor.

### Encephalitis Lethargica (*Encephalitis Epidemica*)

*Definition.* A disease characterised by focal lesions in the grey matter of the brain and central nervous system, often with a disturbance of the sleep mechanism.

*Etiology.* The causative agent is believed to be a filtrable virus, the infection spreading from the naso-pharynx to the arachnoid sac and so to the brain. Encephalitis lethargica occurs in epidemics, but sporadic cases are met with. The disease was noted in Vienna at the end of 1916 and described by von Economo. Early in 1918 it appeared in London, and was at first mistaken for botulism. The virus may be wind-borne, but direct transmission from person to person is uncommon. Epidemics of encephalitis occurred in Japan from 1912 to 1929, and in the St. Louis area, U.S.A., in the late summer of 1933. *Predisposing causes:* 1. Age: Usually between 15 and 45 years. 2. Sex: There is no distinction. 3. Season: Epidemics are liable to begin early in the winter. 4. Locality: Towns especially. 5. Heredity: The disease may be transmitted directly by a pregnant mother to the infant who develops it a few days after birth.

*Pathology.* Encephalitis lethargica has been reproduced in monkeys by subdural inoculation of a filtered extract of infected nervous tissue. The virus produces changes in the grey matter of the brain, especially



in the mesencephalon, basal ganglia and substantia nigra. Thus the cranial nerve nuclei may be affected and less frequently the anterior horn cells in the cord. Sleep regulation is believed to be disturbed when the grey matter between the diencephalon and mesencephalon is affected. At autopsy the brain often presents no naked eye changes, but the leptomeninges may be hyperemic and oedematous, and on section the cortex and the basal ganglia may appear hyperemic, and actual hæmorrhages are sometimes seen in the grey matter. Microscopically changes are found in the various masses of grey matter mentioned above. There is a "cuffing" of the small vessels in the grey matter, due to an infiltration of lymphocytes and plasma cells in the perivascular spaces. Nerve ganglion cells may also undergo destruction in localised patches of grey matter. The cerebral cortex is not usually affected.

**Incubation Period** This is probably about 8 to 10 days.

**Clinical Findings** Certain types of the disease are described. The onset in epidemics is usually acute, sporadic cases may start more insidiously.

**Acute Cases** During the prodromal stage, lasting a few days, the patient may complain of headache, slight sore throat, giddiness, shivering, malaise and occasional vomiting. There may also be diplopia. The temperature varies between 98.6° and 100.4° F. One of three main types may now develop, which will be described separately.

1 *The Somnolent ophthalmoplegic Type* After the prodromal symptoms the patient becomes drowsy.

**On Examination** The patient may be noticed to fall asleep while he is eating and later to become stuporose or delirious. There may be no fever or a low continuous or irregular one of about 99° or 100° F., with at times a rise to 104° F. A rash may rarely be seen on the body, macular, papular or urticarial. The cranial nerves: I Usually normal. II Optic neuritis rarely occurs. III, IV and VI There is commonly ptosis, and external oculo motor pareses occur which may be bilateral. There may be spontaneous nystagmus. The pupils usually react normally, but an Argyll Robertson pupil may be present. V The corneal reflex may be abolished. VII There may be slight facial weakness. VIII There may be vestibular giddiness. IX and X These may be affected. XI and XII Usually these are unaffected. In addition, there may be a lesion affecting the phrenic nerve. In some instances there is evidence of involvement of anterior horn cells, as shown by weakness and hypotonia of muscles in the arm or leg. Upper motor neurone lesions, due to involvement of the internal capsule, secondary to the lesions in the adjacent grey matter may result in stiffness of the limbs or an extensor plantar response. The patient may have aphasia, or at times epileptiform convulsions, and retention of urine or feces may occur. The blood. The red cells show no definite changes. There is usually a leucocytosis of about 25,000 cells per c mm. The Wassermann reaction is negative. The urine. There may be polyuria or glycosuria. The cerebro-spinal fluid. This may be normal, or under increased pressure. It is usually clear. The cells may be slightly

increased, about 20 lymphocytes per c.mm. The sugar content is usually slightly increased, as is also the protein. The Wassermann reaction is negative, and the Lange test may show a luetic curve (see Fig. 25).

2. *The Hyperkinetic Type.* The prodromal symptoms are often marked, with severe headache, pains in the back and vomiting. Pain may be located to one area of the abdomen, such as the appendix region, or to the face, arms or legs.

*On Examination:* The patient is very ill, pale, weak, and the face may be swollen with extensive labial herpes. The characteristic feature is the muscular twitching, which may affect bundles of muscle fibres (fascicular), muscles as a whole (myoclonic), or groups of muscles (convulsive). The patient is dazed, but restless; he is continuously throwing off the bed-clothes, getting out of bed, making noises with his tongue, and his speech is rambling. There may be insomnia, or sleep inversion, the patient being drowsy by day and very restless at night. The pupils are usually small, unequal and react sluggishly or not at all to light or accommodation. The abdominal wall may show rapid myoclonic twitches, the umbilicus being jerked about in varying directions. The diaphragm may be affected, producing hiccough. These myoclonic contractions often persist during sleep. Bilateral choreic movements of a very violent nature may also occur, and the patient must be prevented from self-injury. In some cases athetoid movements are also seen. The deep reflexes are usually normal, but the tone of the muscles is diminished. The patient may later become very still (akinesia), and Parkinsonism supervene.

3. *The Amystatic-akinetic Type.* Here the prodromal stage may be comparatively mild, but the patient notices progressive weakness, and ultimately is confined to bed.

*On Examination:* The characteristic features are the rigidity and the absence of movement, an acute form of Parkinsonism. The patient performs all movements very slowly. There is an increase of muscular tone, resistance being felt when the limbs are flexed or extended. The deep reflexes are not increased. If the patient is up, propulsion and retropulsion (see p. 340) can be demonstrated. There may also be tremors, sialorrhœa, and a greasy skin, due to increased sebaceous secretion. Somnolence or sleep inversion may be present. Further, various oculo-motor or bulbar paræses may be noted, with disturbance of speech or swallowing. The temperature curve is usually only a little above normal.

Certain minor forms of the acute disease will be enumerated, such as :

4. *The apoplectiform type.* The patient is suddenly stricken, as by a cerebral hæmorrhage and may die at once. 5. *The cerebellar type,* with marked ataxia. 6. *The acute bulbar type,* with disturbance of speech and swallowing. 7. *The acute neuritic type,* with paræsis of the face and limbs. 8. *The visceral type,* with abdominal crises resembling those of tabes dorsalis. 9. *The monosymptomatic type.* Hiccough may last 5 to 7 days, or there may be trismus or yawning. 10. *The pseudo-tabetic or pseudo-paralytic type,* with Argyll-Robertson pupils and absent ankle and

knee jerks 11 *The meningitic type*, with symptoms of meningism  
 12 *The psychotic type*, the initial acute delirium suggesting a psychosis

The St Louis epidemic of encephalitis of 1933 was characterised by a sudden onset, with fever, stiff neck, headache, drowsiness and mental confusion. Recovery was rapid, the illness lasting about 2 weeks, although there was a 20% mortality rate. There were no Parkinsonian sequelæ. A filterable virus was isolated.

*Chronic Cases* The manifestations of the chronic form of the disease may directly follow an acute attack, or they may only appear after an interval of several years. In some instances the acute stage of the disease was so slight that it was not recognised as anything beyond a mild feverish illness or a temporary diplopia. The symptoms of the chronic disease are due to destruction of nerve cells, and take the form either of Parkinsonism or of juvenile pseudo psychopathia. *Parkinsonism* Both children and adults are affected. The appearance of the patient resembles that described for paralysis agitans (see p. 340), but the tremors and pill rolling movements of the hands are not often seen. The skin is typically greasy and salivation may be very distressing. The patient usually becomes progressively worse each winter, but in a few cases the disease does not progress. Tonic eye fits or oculogyric crises occur in some cases. The eyes usually are directed upwards and sideways with convulsive movements. Various tics or choreiform movements may also be a manifestation of the chronic disease and in some patients there is a tendency to obesity or polydipsia and polyuria.

*Juvenile Pseudo psychopathia* is another very distressing condition, the child is demoralised, although his intelligence is good. He will commit every manner of evil, such as lying, stealing, and acts of violence. Although he often knows he is doing wrong, he is powerless to control himself. He is often particularly excited at night, and has been described as the "apache" type of child.

*Differential Diagnosis* As there is no specific test for the disease, and as it exists in so many different forms, a diagnosis can only be made by appreciating the nature and possible sites of the lesion and the various clinical pictures which may result. Other conditions which require exclusion are polioencephalitis, botulism, drug poisoning, uræmia meningitis, cerebral tumour, cerebellar lesions, typhus or typhoid fevers, post-vaccinal or measles encephalitis, influenza and paralysis agitans. The cerebro spinal fluid should be examined in every case. In botulism the pupils are usually dilated, the mouth is dry, and there are digestive disturbances. Polioencephalitis (see p. 408). In acute cases, which are rapidly fatal, it is usually impossible to make a diagnosis during life. The young age incidence of the Parkinsonian syndrome is of great diagnostic value.

*Course and Complications.* The course depends upon the severity of the disease. The acute stage may be mild and transitory, but followed later, perhaps at an interval of 4 to 9 years, by Parkinsonism. A severe case may rapidly improve, or relapses ensue during a prolonged illness, or the case insidiously passes into a chronic stage. The important sequelæ are the Parkinsonism and the pseudo psychopathia (see above).

**Prognosis.** The mortality varies in different epidemics, but the average figures show a death rate of 40%, 30% become chronic cases and 30% recover completely. The hyperkinetic type is the most fatal. Death usually results from bulbar paralysis, cachexia, intercurrent infection, or pneumonia.

**Treatment. Prophylactic.** During an epidemic it is advisable to gargle night and morning with a 1 in 1,000 solution of potassium permanganate.

**Curative.** Acute cases should be isolated in bed. Intravenous injections of iodine should be given, using Klemperer's solution (10% sodium iodide in water). A test dose of 20 mils should be injected, and subsequently, if there is no idiosyncrasy, as shown by symptoms of iodism, 50 mils are injected 3 times a week for 3 or 4 weeks. If intravenous injections are not practicable, 2 mils of Lipiodol or Neo-Hydriol (ol. iodisat. B.P. Add.) should be injected intramuscularly 3 times a week for 5 weeks. At the same time hexamine gr. 10 t.d.s. should be given by mouth. If the patient is unconscious, a lumbar puncture should be performed, and the fluid allowed to escape slowly, if it is under increased tension. Sedatives will be required for the hyperkinetic type of disease, the best being an injection of hyoscin. hydrobrom. gr. 1/100 once or twice a day. This is also of value in relieving hiccough. In chronic cases a course of intravenous iodine should first be given, with a view to destroying any active infection which may persist. If no benefit results, a course of intravenous injections of sodium cacodylate should be administered, starting with gr.  $\frac{1}{2}$  in m. 10 of distilled water, increasing by gr.  $\frac{1}{2}$  every fourth day until a dose of gr. 5 is given, and then working down the scale again. For the symptoms of Parkinsonism hyoscin. hydrobrom. gr. 1/100 is injected once or twice daily, or tincture of stramonium may be given by mouth in large doses. It has been shown that fl. oz.  $\frac{3}{4}$  of the tincture produces results comparable with an injection of gr. 1/100 of hyoscin. hydrobromide. If the stramonium causes dryness of the mouth or paralysis of accommodation, pilocarpine nitrate gr. 1/10 can be added to one or more doses of the mixture daily. Hyoscin. hydrobromide gr. 1/50 tablet may be given by mouth, t.d.s., if it is not possible for the drug to be injected. For diurnal somnolence ephedrine hydrochloride gr.  $\frac{1}{2}$  should be given at 8 a.m., and for nocturnal insomnia Medinal (barbitonum sol. B.P.) gr. 3 may be prescribed at night. If sialorrhoea is a very troublesome feature, the parotid region may be exposed to radiation by X-rays. For oculo-epidemic crises Prominal (phenitoinum B.P. Add.), gr. 3 tablet, may be used. One, or one and a half tablets daily may lessen the frequency of the attacks. Benzedrine sulphate (amphetaminum sulphas) mg. 5 tablets by mouth, 4 to 12 daily, may relieve the fatigue, depression and oculo-epidemic crises. It should be administered with caution, and not given if there is arteriosclerosis. Children suffering from pseudo-psychopathia are best treated in special institutions. In some cases improvement is noted with the administration of thyroideum, beginning with gr.  $\frac{1}{2}$  t.d.s., and gradually increasing the dose.

**Acute Disseminated Encephalomyelitis**

**Definition.** Inflammation affecting the brain and spinal cord.

**Etiology.** Certain types are described. 1. Post-vaccinal A complication of vaccination against small pox. 2 After infectious fevers, such as measles, chicken pox, mumps, small-pox, scarlet fever, diphtheria and whooping cough 3 Spontaneous, with no known cause. 4. Other types These include polioencephalitis (see p 400), encephalitis lethargica (see p 321), and certain fulminating cases of meningococcal origin

Acute encephalitis may occur as a complication in the treatment of syphilis with salvarsan compounds, in cerebral syphilis, in encephalitis periaxialis diffusa, in suppurative encephalitis (brain abscess) and locally due to trauma of the skull, or to concussion from a shell explosion.

**Pathology.** The surface of the brain may appear congested, and on section punctate hæmorrhages may be seen Microscopically, there is a diffuse infiltration of the perivascular spaces with round cells and plasma cells, and demyelination of the white matter may be present around the vessels The pons, medulla and lumbar part of the cord are especially liable to be affected

**Clinical Findings.** 1 *Post vaccinal Encephalomyelitis.* This was first described in 1912 (see also p 550) The symptoms usually begin 10 to 12 days after a primary vaccination in a child or adult The patient complains of malaise, headache and vomiting The temperature rises to over 102° F, the legs become paralysed, and the plantar responses may be extensor There is later incontinence of urine and feces in cases which die in coma The patient may recover completely, or die in a day or so, or recovery may be incomplete with residual states, such as hemiplegia

2 *Encephalomyelitis associated with Infectious Fevers* The symptoms occur as a complication of measles, mumps, and chicken pox, the legs may rapidly become paralysed, and there is retention of urine. The paralysis may be flaccid with diminished reflexes and later spastic with increased reflexes, and an extensor response. There may also be dissociated anæsthesia of the legs, pain and temperature sense being lost, and touch vibration and postural sense being unaffected. In other cases there are convulsions with headache. The cerebro-spinal fluid This is often normal, but in some cases there is an excess of cells and protein

3 *Spontaneous Encephalomyelitis* The patient is usually a young adult who is suddenly taken ill, either with pains in the body or limbs, or with weakness of the legs

**On Examination** There is often a spastic paresis or paraplegia, with exaggerated or lost deep reflexes, and usually an extensor plantar response There may be sensory loss in one or both legs, or a Brown-Séquard syndrome with paralysis of one leg and loss of sensation on the other side. There is usually retention of urine The cranial nerves are seldom affected, but there is often nystagmus In some cases meningeal symptoms are present, or there may be aphasia and hemiplegia The cerebro-spinal fluid is normal.

**Differential Diagnosis.** The disease is probably distinct from acute disseminated sclerosis. Thus the fever, shooting pains, loss of pain and temperature sensations, the normal cerebro-spinal fluid, and at times loss of deep reflexes in the legs during the acute stage, are in favour of encephalomyelitis. In acute disseminated sclerosis there is more likely to be diplopia, loss of postural and vibration sensations in the leg, and retrobulbar neuritis. Other conditions which may require exclusion are polioencephalitis (see p. 406), encephalitis lethargica (see p. 321), and meningitis (see p. 288).

**Course and Complications.** The disease may pursue a rapidly fatal course, or there may be recovery, after an acute illness lasting 1 or 2 weeks. Sequelæ include a persistent Brown-Séquard syndrome, a condition resembling chronic disseminated sclerosis, hemiplegia or aphasia.

**Prognosis.** Only a few cases die; the majority recover.

**Treatment.** The patient must be kept in bed and symptomatic treatment applied. In post-vaccinal encephalomyelitis 5 to 30 mls of serum, obtained from an individual successfully vaccinated 14 days previously, should be injected intravenously, or into the patient's theca after lumbar puncture.

### Encephalitis Periaxialis Diffusa

#### (Schilder's Disease)

**Definition.** A disease characterised by inflammatory changes in the white matter of the brain, with demyelination of nerve fibres.

**Etiology.** This is a rare disease, the cause of which is unknown. Children and young adults are chiefly affected.

**Pathology.** The brain often appears small. On section, sharply defined patches of greyish or reddish-brown hyaline appearance are seen in the white matter. The occipital and temporal lobes are especially likely to be affected. Lesions also occur in other parts of the cerebrum, and these may coalesce. The cerebellum is sometimes affected. Microscopically, it is seen that the myelin of the white matter is destroyed, with secondary degeneration of the axis cylinders. An infiltration of round cells and neuroglial cells is present in the white matter, and phagocytes and lymphocytes are gathered around the vessels.

**Clinical Findings.** The patient is often a child about the age of 8 or 10 years. The symptoms depend upon the area of brain affected; in a typical case the patient is comparatively suddenly taken ill with malaise and headache. There may be early disturbance of vision, giddiness, deafness, or difficulty in walking, or in using an arm.

**On Examination:** If the cortical motor area is involved there may be spastic weakness of a leg or arm, with exaggerated deep reflexes and an extensor plantar response. With involvement of the occipital cortex there is dimness or loss of vision. Thus both visual fields may be totally blind, or with unilateral lesions there is a homonymous hemianopia. The pupils react normally, and the discs are normal; occasionally there is a slight degree of optic neuritis. When the frontal lobes are affected

there may be alteration in mentality, such as childishness or actual dementia. In other cases there is disturbance of speech or aphasia. Jacksonian fits or generalised epileptiform convulsions may occur. The cerebro spinal fluid. This is normal. The temperature is usually normal, but may rise before death.

**Differential Diagnosis.** The diffusely scattered and spreading nature of the lesions usually serves to differentiate the disease from a cerebral tumour. With the latter, papilloedema is generally more intense.

**Course and Complications.** The course is usually steadily progressive, arrest being very rare.

**Prognosis.** The disease is usually fatal either in a few months or in 2 to 3 years.

**Treatment.** No cure is known. The administration of iodides or of arsenic is unavailing.

### Cerebral Diplegias

(including *Little's Disease*)

**Definition.** A disease of infants characterised by spastic diplegia, or spastic rigidity of the arms, trunk and legs.

**Etiology.** In *Little's disease* the causes are operative before or during birth. They include birth injuries resulting in cerebral venous hæmorrhage, hæmorrhagic disease of the new born, intra uterine infections and faulty development (agenesis) of the pyramidal tracts. In other cases of cerebral diplegia the spasticity develops in early life, often due to encephalitis and rarely to hypothyroidism. Infantile hemiplegia may be due to hæmorrhage, infections and possibly to degenerations.

**Pathology.** Cerebral venous hæmorrhage, or petechial and confluent hæmorrhages may be found post mortem. In other cases the convolutions of the brain appear atrophied on naked eye examination, and microscopically atrophy or sclerosis of the pyramidal tracts is seen. Multiple small cysts (porencephaly) are at times present in the brain.

**Clinical Findings.** No symptoms are usually noticed at birth. The infant does not begin to walk until after the normal age (1 year). It is then noticed that the legs are weak.

**On Examination.** There is spasticity of the legs, with an increase of the deep reflexes. The extensor plantar response of the infant is perpetuated. Later the heels become raised, and the child cannot walk on them, the legs are adducted, and may actually cross one another on walking (scissors gait). The arms are rarely affected. In the majority of cases the mentality of the child is normal. In other cases mental deficiency, athetoid movements or epileptiform fits may occur. There is usually a tendency to improvement, but death may result from some intercurrent disease.

**Treatment.** In the early stages massage and passive movements should be applied to the affected limbs. Later contractures may be improved by operations such as tenotomy. Active remedial exercises

are useful in the co-ordination of movements. The administration of thyroideum in doses of gr. 1/10 t.d.s. is of value in cases associated with hypothyroidism.

### Amaurotic Family Idiocy

#### (Cerebro-macular Degeneration. Tay-Sachs Disease)

**Etiology.** The cause is unknown. The patient is nearly always a Jewish infant. It tends to affect several members of one family.

**Pathology.** There is degeneration of nerve cells, with deposition of lipid material throughout the brain and cord. The nerve cells swell and the nuclei are destroyed.

**Clinical Findings.** The infant appears healthy at birth, but between the age of 3 and 6 months muscular weakness is noticed. This often starts in the back, and becomes generalised. Ophthalmoscopic examination shows a condition of primary optic atrophy, with a cherry red spot at the macula due to atrophy of the retina and exposure of the choroid. The disease is progressive, there is marked mental deterioration, and death occurs in about 6 to 12 months from the onset of symptoms.

**The Juvenile Type.** Another type of the disease affects young children, usually not Jews. There is some degree of optic atrophy with retinitis pigmentosa, but the red spot does not appear at the macula.

### Epilepsy

#### (Idiopathic Epilepsy)

**Definition.** A chronic disorder in which there are recurrent attacks of unconsciousness with or without convulsions, due to no known cause.

**Etiology.** In some cases an attack may be provoked by a fright, but air raids have not increased the incidence of fits in epileptics. **Predisposing causes:** 1. Age: The majority of cases begin in childhood or about the time of puberty. In a few cases the attacks first appear after the age of 50. 2. Sex: The incidence is equal. 3. Hereditary transmission: This occurs in a definite proportion of cases. 4. Neuro-pathic disposition: This is probably an important factor. 5. Rickets and infantile convulsions. 6. Dyspituitarism: Especially of the Frohlich type. 7. Migraine may be a precursor. 8. Left-handed individuals are unduly susceptible. 9. Local cerebral lesions are present in a certain proportion of cases with generalised fits.

**Pathology.** The following theories have been suggested to explain the manifestations of epilepsy: 1. Irritation of the cerebral cortex. 2. Inhibition of the cerebral cortex. The convulsions are due to uncontrolled stimuli from the lower cerebral centres (a "release" phenomenon). 3. Anaphylactic shock. The fall in blood pressure and leucopenia favour this view. 4. A metabolic dyscrasia. Some unknown epileptogenous agent circulates in the blood from time to time. Thus the serum taken from a patient during an attack may induce convulsions on injection into an animal, whereas the serum between the attacks is innocuous. The chemistry of the blood and cerebro-spinal fluid shows no abnormalities between or during the



attacks The attacks are apt to remit during pregnancy and to be intensified in relation to menstruation, this may be due to some metabolic disturbance 5 As the electro encephalogram shows that epilepsy is associated with the development of abnormal rhythms in the cerebral cortex, it has been called a paroxysmal cerebral dysrhythmia

It therefore appears likely that two factors are required, a congenital or acquired cerebral defect and an unknown toxic agent In organic epilepsy there may be any form of cerebral lesion but convulsions only occur in a certain proportion Thus with cerebral tumours epileptiform convulsions are noted in less than 10% of cases Post mortem, no changes are found in the central nervous system of men or animals who have suffered from idiopathic epilepsy.

**Clinical Findings** The patient is usually a child or young adult There may be a history of infantile convulsions The general health is often good, but the patient is apt to be lazy, egocentric and prone to lying The speech may be of the "plateau" type, the vowels being pronounced with a peculiar even tone The clinical findings of idiopathic and organic epilepsy are indistinguishable, and the so called Jacksonian, or local fits, are met with at times in many cases of idiopathic epilepsy in which there is no local lesion of the brain Certain types of epilepsy are described —

1 *Petit mal* (minor attacks) The patient may be quite unaware of their occurrence The onlooker notices that the patient suddenly stops speaking or remains motionless for a second or so He looks dazed, the pupils may dilate, the head moves slightly, the eyes deviate and there may be yawning The face usually becomes pale and subsequently flushes The patient then comes round suddenly and continues what he was doing During the attack there is a brief loss of consciousness In other cases the patient may suddenly fall to the ground without warning (*morbus caducens*), he gets up again at once after a momentary loss of consciousness Another type of minor attack is characterised by what is known as a "psychic equivalent" in which the patient may, for example, become a homicidal maniac or perform automatic actions of which he is unconscious

2 *Grand mal* (major attacks) In about half the cases there is a preliminary phase known as an aura *The aura* This may take the form of auditory, visual, olfactory or gustatory sensations In other cases numbness or tingling may be felt in one of the extremities, or a sensation of something travelling up the arm or leg There may be slight muscular twitchings, cramp, sweating, giddiness, or peculiar dreamy states or reminiscent sensations of familiarity with certain places with which the patient is personally unacquainted These auras indicate the beginning of a fit in some portion of the brain and the process, whether it be one of excitation or inhibition, spreads rapidly, producing unconsciousness and convulsions These usually affect first such muscles as those which move the head and eyes and then rapidly become generalised The patient may give an initial unconscious cry and then fall to the ground There is marked pallor *The tonic phase* This lasts for about half a minute The muscles are contracted

and the attitude is often that of decerebrate rigidity, the arms being flexed and adducted, the legs extended, the head retracted and often turned to one side, and the back arched. Respiration usually ceases with resultant cyanosis. The tongue is protruded and bitten as a result of masticatory spasm. *The clonic phase.* This is characterised by jactitations, violent muscular contractions and relaxations producing convulsions. They may be first noted on one side of the body. Breathing is stertorous and frothy fluid exudes from the mouth, which is blood-stained if the cheeks or tongue have been bitten. The face is contorted, the mouth opening and closing and the eyes rolling upwards. There is frequently enuresis, and the pulse is rapid and of small volume. The pupils are dilated and do not respond to light, the corneal and deep reflexes are usually abolished and the plantar responses are often extensor. It is believed that the blood pressure falls during an attack. After 3 or 4 minutes the convulsions cease, and the patient falls asleep or rapidly regains consciousness. He does not remember the attack, but may be made aware of it by finding that he has injured himself or that he is in some unusual position, or that he has had incontinence of urine. On recovering consciousness he may complain of severe headache or of nausea or vomiting. There is frequently polyuria with a trace of albumin in the urine, and an extensor response may persist for a few hours, with increase of the deep reflexes. In some cases there is a temporary aphasia or post-epileptic paralysis (Todd's paralysis). Changes in the electro-encephalogram have been found in 50% of patients under the age of 40 years, suffering from convulsive epilepsy, even although no fit is seen at the time of the examination. The abnormality between the fits consists in the occurrence of intermittent, irregular slow waves. "Seizure" waves occur during a fit; in grand mal these are sharp spikes, in petit mal quick sharp spikes alternate with slow round waves, and in psycho-motor attacks (psychic variants) square flat waves are seen (see Fig. 24).

3. *Status epilepticus.* The patient may pass into a state of coma interrupted by convulsive attacks without recovery of consciousness. The temperature is raised to 104° or 105° F., the pulse is frequent and of small volume, there is rapid bodily wasting and death is likely to occur in a few days from fatty myocardial degeneration or pulmonary oedema.

Special Varieties of Epilepsy. 1. *Cursive Epilepsy.* The patient suddenly rushes about, forwards, backwards, or from side to side, being unaware of his actions.

2. *Pyknolepsy* (Friedmann-Heilbronner disease). This is a disease of children closely resembling petit mal. The onset is usually between the ages of 4 and 12 years. The attacks begin quite suddenly and are of very brief duration, lasting only a few seconds. The child has a vacant look and is unable to speak or move. There may be twitching of the eyelids, and the eyes may roll upwards. There are no convulsions and the child does not fall and is not really unconscious. Recovery is sudden and complete. As many as 100 such fits may occur in a day. The attacks usually disappear after puberty and are totally uninfluenced by drug treatment.

**3 Myoclonic Epilepsy** This type is characterised by excessive muscular contractions of the myoclonic type (see p 323)

**4 The Lenticular Type** There is marked rigidity of the trunk and limbs, speech is blurred and slow, and facial contractions may be noted

**Differential Diagnosis** Idiopathic epilepsy must be diagnosed from 1 **Infantile convulsions** Clinically the convulsive phenomena are alike, and epilepsy can only be excluded by the course of the disease, infantile convulsions being of a temporary nature, epilepsy tending to recur 2 **Jacksonian epilepsy** This type is in some cases organic and associated with a definite cerebral lesion such as a depressed fracture of the skull tumour of the brain gunshot wound of the head, etc Iocil fits ensue In other cases local fits occur in idiopathic epilepsy 3 **Epileptiform convulsions associated with uræmia, congenital cystic disease of the kidney alcoholism, lead poisoning, cerebral syphilis, general paralysis of the insane, cerebral cysticercosis, etc** 4 **Hysterical attacks** During these attacks the patient is not completely unconscious The attacks occur in the presence of onlookers, and the patient does not fall in a dangerous situation The corneal, pupil and deep reflexes are usually present The patient does not bite his tongue nor micturate Hysterical attacks may alternate with epileptic ones 5 **Syncopal attacks** These are associated with a fall in blood pressure and are usually provoked by some unpleasant sight or thought There is loss of consciousness and convulsions may occur Adams Stokes' attacks are associated with cerebral anæmia (see p 220) 6 **Vasovagal attacks (medullary syndromes)** These resemble a much prolonged attack of petit mal There is cutaneous pallor and sweating, a slow and feeble pulse dilated pupils flaccid muscles abdominal, cardiac or respiratory discomfort and the patient may fall 7 **Narcolepsy** This may be differentiated from epilepsy Males are affected slightly more frequently than females usually about the age of puberty or more rarely about the age of 40 and the disease is very obstinate It is characterised either by sudden attacks of uncontrollable sleep, or by the cataplectic attacks with loss of muscle tone The latter are induced by emotions especially by amusement The patient falls but retains consciousness He is unable to speak or to move the lower jaw drops and the eyelids tend to close After a few seconds or minutes he suddenly recovers There may be protrusions of the tongue or contraction of the facial muscles possibly due to the patient trying to speak The patient usually describes the attack by saying that he 'flops' In some cases narcolepsy is complicated by epilepsy Traumatic cases also occur following blows on the head and possibly result from minute hæmorrhages near to or in the third ventricle

**Course and Complications** The course of epilepsy is very variable Minor attacks may occur frequently and be interspersed with major fits Major attacks may recur at comparatively long intervals Complications include post-epileptic phenomena, such as automatic actions Thus the patient may undress after an attack, being quite unaware of his actions Mania and paralysis may ensue Rupture of the heart, and

fracture or dislocation of the jaw, may occur during the convulsive stage. Mental deterioration is especially likely to follow attacks which begin early in childhood; it does not depend upon the violence of the attacks.

**Prognosis.** The outlook is usually unfavourable in epilepsy, and spontaneous recovery is comparatively infrequent. The most hopeful signs are the absence of minor attacks and long spaced-out major attacks. Petit mal is less easily influenced by treatment. Status epilepticus is a very grave event; death may be due to an accident, such as suffocation during a nocturnal attack.

**Treatment.** *Prophylactic.* An attack may sometimes be aborted when there is an aura of something travelling up the limb, by tying a bandage around the limb close to the trunk. Firm muscular contraction by clenching the fist may at times avert an attack heralded by a recognised aura. In view of the hereditary transmission of a neuropathic nature it is inadvisable for epileptics to marry.

*Curative:* 1. During the attack. No special treatment is usually required for attacks of petit mal. The immediate treatment for an attack of grand mal consists in loosening the patient's collar, removing false teeth if present, and preventing him from biting his tongue by inserting a spoon between his jaws. The patient should not be aroused if he falls asleep after the attack. If there is much collapse a hypodermic injection of a stimulant such as strychnin. hydrochlor. gr. 1/60 may be given. A patient who is subject to attacks of epilepsy occurring by night should sleep on a low bed.

2. Between the attacks. Treatment is best commenced by the administration of potassium or sodium bromide. If the attacks occur by night gr. 20 should be given each evening before retiring to bed. If the attacks occur by day a dose of gr. 10 may be given twice a day or gr. 20 in the morning. The addition of liq. arsenicalis m. 2 or 3 will usually prevent the appearance of a bromide rash. Over-dosage of bromides is shown by a furred tongue, drowsiness and slowness of speech. If the bromide controls the fits adequately it should be taken regularly and continued for at least 3 years after the last fit. If the bromide is not successful a trial should be given to Luminal (phenobarbitonum B.P.), beginning with a dose of gr.  $\frac{1}{2}$  once or twice a day and continuing with the bromide as before. Luminal is more efficacious than the soluble sodium salt. The Luminal may be increased up to gr. 1 twice a day. In some cases it is very efficacious in controlling the fits, but there are certain drawbacks to its use; thus the patient may rapidly become tolerant, necessitating an increase of the dose, or the drug may produce toxic effects, such as dementia or delirium after even a small dose. In some cases the fits have increased in number or severity after the Luminal has been discontinued. For the minor attacks characterised by falling, a dose of Luminal gr.  $\frac{1}{2}$  taken regularly at night is of value. Other drugs which are sometimes used include tincture of belladonna m. 7 to 15. This may control the attacks when other drugs fail. Sodium biphosphate gr. 10 to 15 can be used with a bromide mixture, and aids in controlling both major and minor attacks. The addition of tincture of digitalis m. 5 to 10, to a bromide mixture,

is of value in some cases. Sodium diphenyl-hydantionate (Epanutin Dilantin) was introduced in 1938 for the treatment of epilepsy. It is put up in capsules containing gr  $1\frac{1}{2}$  for adults and gr  $\frac{3}{4}$  for infants. It is a powerful anti convulsant, but not without danger. The patient must be under very careful medical supervision during the first few weeks of its administration. It should only be tried when other drugs such as bromides, Luminal, belladonna and borax have failed. If Epanutin then fails it should be given in combination with Luminal or bromide. It is more efficacious in grand mal than in petit mal. Toxic symptoms include nausea, vomiting, ataxia, tremors, headache, hallucinations, delusions and cutaneous rashes. Epanutin should be discontinued at once if any of these occur. The change over from bromides or Luminal should be made slowly. Thus if the patient was taking three doses of Luminal daily, one dose of Luminal should be replaced by one capsule of Epanutin each week. It may be advisable to continue the administration of one dose of Luminal with 3 to 5 capsules of Epanutin daily. Similarly the change back from Epanutin to other drugs should be made gradually or status epilepticus may be precipitated. In all cases a daily action of the bowels should be ensured. For status epilepticus the patient should be kept in bed in a dark room, and an immediate subcutaneous injection given of morphin sulph gr  $\frac{1}{4}$  or hyoscin hydrobrom gr  $1/200$ . A subcutaneous injection of Luminal Sodium gr 2 in 11 of water should then be given and if this does not stop the convulsions an enema may be administered, followed by a rectal injection of  $\frac{3}{4}$  to 1 fl oz of paraldehyde (for an adult) with an equal quantity of olive oil, and the temperature may be lowered by tepid sponging.

*General Treatment* The patient should be encouraged to take an interest in outside affairs as far as possible, but mental worry and over-fatigue are injurious. A regular mode of living, with open air exercise and avoidance of alcohol should be enforced. No epileptic should be allowed to drive a car or engage in any occupation in which he may be a danger to himself or to others. Children of the poorer class are often best treated in special institutions or colonies.

*Diet* It is now thought that a salt poor diet has no special merit in the treatment of epilepsy. A ketogenic diet is of temporary value in some cases, especially in children. The method which was described in previous editions is now, however, seldom used.

*Narcolepsy* This may be treated on the same lines as epilepsy, in some cases good results have been obtained by the administration of ephedrine hydrochloride gr  $\frac{3}{4}$  three times a day for an adult, and gr  $\frac{3}{4}$  for a child, or by Benzedrine sulphate (amphetamina sulphas), mg 5 tablets, 2 to 18 daily.

### Chorea

(Sydenham's Chorea Rheumatic Chorea St. Vitus' Dance)

*Definition.* A disease characterised by spontaneous and irregular muscular contractions, usually associated with an acute rheumatic infection.

**Etiology.** The cause is not definitely known, but in the majority of cases chorea is believed to be due to a rheumatic infection. *Exciting causes:* 1. *Fright.* 2. *Pregnancy, especially during the first three months of the first pregnancy.* *Predisposing causes:* 1. *Age:* Childhood and adolescence, rare after 25. 2. *Sex:* Females predominate. 3. *Overwork at school.* 4. *Poverty and unhygienic surroundings.* 5. *A familial rheumatic tendency.*

**Pathology.** It is believed that the essential change consists in a meningo-encephalitis in which the pia-arachnoid, cerebral cortex, the caudate nucleus, the red nucleus and possibly the superior cerebellar peduncles are affected. A focal lesion of the corpus Luysii (subthalamic body) will cause hemichorea on the opposite side. The meningo-encephalitis may be due to infection by the *Diplococcus rheumaticus* of Poynton and Paine. *Previously chorea was considered to be due to minute cerebral emboli, associated with rheumatic carditis, or to cerebral thromboses.*

**Clinical Findings.** The patient is often a child of about the age of 10. The child becomes nervous, irritable and unnaturally emotional. She is listless, inattentive and has difficulty with her lessons. There may have been loss of appetite, and sore throat, growing pains or rashes on the skin (such as erythema nodosum). In some cases the child seems lame or drags one leg, or is clumsy, and is apt to drop things, and she may have difficulty in speech when excited. Twitching of the face, grimacing, twitching of the hands, or shrugging the shoulders may be the first sign noticed in other instances.

**On Examination:** The signs present vary with the severity of the case.

**A Mild Case:** The twitching movements are seen chiefly on both sides of the face, and in one hand, arm or shoulder. Tremors are seen when the arm is extended and there is a tendency for the arm on the affected side to droop, with flexion of the wrist and over-extension of the metacarpo-phalangeal joints. The tongue may be jerked in and out when the patient is asked to protrude it. Jerky movements may occur at the elbow and shoulder when the patient grips the examiner's hand. The temperature is normal, but the heart may be slightly dilated.

**A Moderate Case:** The movements now are very obvious and the child is hardly ever still. The legs are less affected than the arms. When only two limbs are involved they are always homolateral. The face and trunk muscles are bilaterally affected. The movements are described as spontaneous, large and irregular. To a certain extent they can be controlled and they cease during sleep. The affected muscles are lacking in tone and the arm does not swing on the affected side on walking. Hypermetria is shown by the difficulty in grasping objects, and by the finger overshooting the mark in the finger-nose test. Dysdiadokokinesis may also be present (see p. 354). The reflexes: The cutaneous reflexes are normal. The deep reflexes are diminished in moderately severe cases, and the knee-jerks are "sustained," the leg being hung up for a brief period before it falls again. The sphincters are not affected. Sensation is normal. The temperature is often

normal, unless the illness is complicated by an active carditis, and the pulse is not so rapid as in rheumatic fever

**Severe Cases (Chorea Gravis)** The movements are violent and the patient may be thrown from a chair or bed. There is disturbance of speech and of deglutition. Maniacal symptoms may appear, or there may be delirium with visual hallucinations. The temperature is usually raised and signs of endocarditis are rarely lacking. Other types described include *Chorea mollis*, in which there is a flaccid paresis of the voluntary muscles with only slight movements. *Paralytic chorea*, one arm or leg may be limp and almost useless.

**Differential Diagnosis** The diagnosis of a typical case of chorea usually presents few difficulties. Certain other conditions may require consideration such as 1 A tic. Here the movements are repetitive and limited to certain muscles. 2 Hysteria. St. Vitus' dance, as originally described, was presumably a hysterical manifestation. The movements in hysteria (see p. 347) have not the character nor distribution of those in chorea. 3 Symptomatic chorea. This occurs in such diseases as epilepsy, infantile cerebral paralysis, tabes dorsalis, general paralysis of the insane, or as a post-encephalitic symptom. 4 Encephalitis lethargica. In the hyperkinetic variety of this disease there is generally a III nerve paralysis (see p. 323). 5 Athetosis. The movements here differ from those of chorea and are usually confined to the hand or arm. They are generally the sequela of a hemiplegia. 6 Huntington's chorea. The age incidence is later, there is a familial history and usually mental degeneration.

**Course and Complications** Mild cases respond to treatment in a few weeks, but if the condition is overlooked or the child is pressed at her work the disease is prolonged. Recurrent attacks occur in about 30% of cases, often after an interval of a year. A watch should be kept for complications such as endocarditis or pericarditis. Aortic disease rarely occurs. Mitral stenosis often develops many years after an attack of chorea.

**Prognosis** This is good, except in chorea gravis and maniacal chorea. In chorea gravis dysphagia is a dangerous symptom, and maniacal chorea is usually rapidly fatal. The gravity of the disease is increased by endocarditis or pericarditis. Abortion is liable to occur in the chorea of pregnancy, and may be followed by the death of the mother.

**Treatment.** In all cases the child should be taken away from school and kept quietly at rest at home, preferably in bed, unless the disease is very mild. In severe cases precautions should be taken to prevent the child injuring herself by falling out of bed. Adequate nourishment is necessary and milk is of value in this respect. The bowels should be kept open daily. Warm baths tend to relieve the nervous symptoms. There is no specific curative drug. Good results are usually obtained with aspirin in doses of gr. 10 t.i.d. for a child of 10 to 12 years. In other cases, liq. arsenicalis may be given in doses of m. 3 to 5, t.i.d. p.c., care being taken that neuritis does not occur. Some physicians claim good results with Chloretone (chlorbutol B.P.) gr. 5 t.i.d. in a

cachet, and this may be given if the child is very restless. In maniacal chorea a mixture containing Pot. brom. gr. 10, chloral hydrat. gr. 15, syr. aurant. m. 30, aquam ad fl. oz.  $\frac{1}{2}$ , should be given t.d.s. for a child of 12, or Avertin (bromethol B.P. Add.) 0.075 mil. per kg. body-weight may be given per rectum. In some cases an injection of hyoscin. hydrobrom. gr. 1/300 is of value. If the convulsions are not checked, an inhalation of chloroform may be necessary from time to time. Fever therapy has been used with good results in some cases in America. The body temperature is raised to 105° or 106° F. by means of a hot box, and is kept at this level for several hours. This is repeated once or twice at intervals of 5 or 7 days. During the illness a careful watch should be kept on the heart to detect signs of dilatation, endocarditis or pericarditis. Should they occur, further rest in bed is required as described on pp. 205, 240. Convalescence should never be hurried, and the child should not be allowed to return to school until all movements have ceased. A tonic containing iron, such as syr. ferri phosph. co. m. 60 t.d.s., p.c., is of value. No examinations should be allowed for a year after returning to school.

### Huntington's Chorea

#### (Chronic Progressive Chorea)

**Definition.** A disease characterised by choreiform movements and mental deterioration.

**Etiology.** The cause is unknown. *Predisposing causes*: 1. Heredity: The disease presents a definite familial incidence, and in some cases the mental and physical characteristics are transmitted independently. In successive generations the disease tends to show itself earlier; further, if one generation escapes, the disease does not reappear. 2. Age: Usually between 30 and 50 years. 3. Sex: The incidence is equal.

**Pathology.** The brain is usually below the average size, with some atrophy of the frontal lobes and corpus striatum, the putamen is especially affected, with secondary involvement of the globus pallidus. The mental changes may be due to atrophy of the cortical nerve cells.

**Clinical Findings.** Choreic movements are first noticed in the face or in the muscles moving the head. The arms and legs are subsequently affected. The speech may have a peculiar explosive character. The oculo-motor muscles are usually the last to be involved. The patient finally becomes chair or bed-ridden. The mental changes are insidious, the patient is irritable and lacking in external interests, and gradually his higher mental faculties fail.

**Differential Diagnosis.** The character of the motor and mental changes, with a familial history, usually establishes the diagnosis. The blood and cerebro-spinal fluid should be examined to exclude neurosyphilis. Senile chorea: The age of onset is later. There are choreic movements, but no mental changes. This may be an atypical form of Huntington's chorea. Congenital chorea: This is present from birth and is probably due to agenesis of the cells in the corpus striatum.



There is no spasticity of the limbs, but the mental processes are rather slow. The condition appears to be related to Huntington's chorea. Apoplectic chorea. This results from a hæmorrhage into the substantia nigra in the mid brain. The patient has choreiform movements and usually dies in a week or so.

**Course and Complications** The course is usually slow, and the patient may live for 20 years or so after the first appearance of symptoms. Homicidal or suicidal mania may occur as complications.

**Prognosis** The disease, although not curable, is not necessarily fatal and death is often due to an intercurrent infection.

**Treatment** *Prophylactic* Marriage should be avoided by a sufferer from the disease.

*Cure* No drug is known to have any effect in checking its course.

### Electric Choreia

(*Dubini's Disease*)

This is probably a myoclonic form of encephalitis lethargica (see p. 823). The patient is very ill with pains in the back, violent muscular contractions and fever. The disease is usually fatal in a few weeks.

### Pink Disease

(*Infantile Acrodynia Erythrædema Trophodermatonecrosis*)

**Definition** A disease of infants characterised by mental disturbances, insomnia, sweating, disordered sensation of the extremities and peripheral vascular phenomena.

**Etiology** The disease is believed to be due to an unknown virus, or to the deficiency of vitamin B<sub>1</sub>.

**Predisposing causes** 1 Age Usually 4 months to 4 years. 2 Sex Equal incidence. 3 Season Winter and spring. 4 Locality Central Europe, Australia and North America. Sporadic cases may occur anywhere. Localised epidemics have been described.

**Pathology** The essential lesion is believed to be an encephalitis affecting the sub-thalamic centres. Peripheral neuritic changes have also been described.

**Clinical Findings** *Prodromal Stage* The onset is insidious, but there is probably an early stage with slight fever lasting a few days. The earliest change noted by the parents is often an alteration in their child's disposition. The infant may become listless, or exhibit fits of temper characterised by screaming and biting. He may complain of itching, tingling or burning of the soles of the feet or palms of the hands. The appetite is often poor and thirst excessive, and micturition may be delayed.

**On Examination** The child avoids the light and may lie curled up, or bury his head in the pillow, or sit forward with his head between his feet. The feet and hands are cold and the soles and palms resemble raw beef in their colour and sodden appearance. An erythematous rash may also be seen on the trunk and limbs in some cases. These skin changes do not always appear, and may not be noted until late in the

disease. Gangrene of the fingers and toes occurs at times. The child may be seen rubbing his feet and hands or placing them on a cold surface or sucking his fingers. There may be profuse sweating, especially of the hands and feet. Muscular weakness and hypotonia with wasting may be a prominent feature, so that the child cannot walk. Myoclonic movements are sometimes seen. The teeth may also fall out. The deep reflexes are diminished. The inguinal, axillary and intercostal glands may be enlarged. Desquamation of the hands and feet occurs in the terminal stages of the disease, before the pink colour disappears. The temperature is usually normal, but a tachycardia of 140 to 180, persisting by day and night for several weeks, is often found. The blood pressure is usually raised to 110 to 130 mm. Hg. The white cells show a leucocytosis of 10,000 to 30,000 per c.mm. Lumbar puncture in the early febrile stage usually reveals a fluid showing meningitic changes with a slight excess of protein and cells. Insomnia may be a very pronounced feature of the disease.

**Differential Diagnosis.** The early stage is usually considered to be due to some slight febrile disorder. The excessive thirst may suggest diabetes insipidus, and the muscular weakness poliomyelitis, post-diphtheritic paralysis or amyotonia congenita. Several cases have been mistaken for tuberculous meningitis. The gangrene of the fingers or toes may suggest Morvan's disease.

**Course and Complications.** The disease may last for a few weeks up to six months, and may be interrupted by remissions or by exacerbations and relapses. Complications include septicæmia and bronchopneumonia.

**Prognosis.** This is usually good and there are no sequelæ. Progressive wasting or intercurrent infection causes death in about 5% of cases.

**Treatment.** The child must be very carefully nursed. A soothing application for the hands and feet consists of a paste made of equal parts of Zinc oxide, calcium carbonate, glycerin and water. When the burning is very intense, the hands can be covered with rubber gloves and placed in ice-cold water. Some sedative drug, such as Luminal (phenobarbitonum B.P.) gr.  $\frac{1}{4}$  for a baby of 4 months and gr.  $\frac{1}{2}$  for an infant of 4 years, should be prescribed for the insomnia. Favourable results have been reported by the administration of vitamin B<sub>1</sub>. This may be given by intramuscular injections of Benerva (aneurin, hydrochlor. B.P.Add.) mg. 2 in 1 mil. three times a week.

### Paralysis Agitans (Parkinson's Disease)

**Definition.** A disease characterised by muscular rigidity and tremors.

**Etiology.** The cause is not known. *Predisposing causes:* 1. Age: Usually after 50. 2. Sex: Males predominate.

**Pathology.** The lesion is considered to lie in the extrapyramidal motor system of the brain, resulting from arteriocalillary fibrosis. The

corpus striatum, especially the caudate nucleus and the putamen, is thought to be affected. The corpus striatum probably controls the tone of muscles. Lesions of the corpus striatum produce rigidity and tremors resulting from the release of lower centres.

**Clinical Findings** The patient first notices some difficulty in performing actions with one hand, and he may be conscious of the muscles being somewhat stiff. He may also feel an aching pain in the arms or back, some general fatigue or perhaps sensations of heat or cold. After a variable time the tremors appear.

**On Examination** *An early case* Stiffness may be found in the muscles of one hand or forearm and the face may be rather expressionless. Slight tremors may be seen in one hand. The leg on the same side is often next affected. Later, the tremors spread to the other hand and the rigidity becomes generalised. *A developed case* In the course of about two years the appearance of the patient is very striking. The face lacks expression, is vacant and somewhat staring and immobile (the Parkinsonian mask). The lower lip may twitch, but the eyelids seldom blink. The forehead is wrinkled or very smooth. The whole attitude is one of flexion, the neck and trunk are slightly flexed. The shoulders are slightly abducted, with the elbows flexed and held away from the body, the hands in front of the abdomen. The hands are held with the metacarpo-phalangeal joints flexed, and the interphalangeal joints extended and the thumb opposed to the index. The hips and knees are slightly flexed. The movements are all stiff and slow owing to general muscular rigidity. On looking to one side, the eyes move before the head, and the head and trunk move together. In walking the gait is shuffling, the steps are rather short and the arms swing very little. Owing to the flexion of the trunk the balance is easily upset. If the patient catches his foot in an object on the floor, or if he is pushed from behind, he hurries forward with short steps as it were to overtake his centre of gravity. This is known as propulsion and the gait is festinant. Similarly, if the patient is pushed backwards or to one side (retropulsion or lateropulsion) he tends to fall down, and to prevent this has to move quickly in the direction in which he has been pushed. The tremors have a peculiar rhythmic character of about 4 to 7 vibrations a second. They are usually most marked in the fingers. The movements of the thumb and index finger produce a pill rolling effect. There may also be movements of flexion and extension or radio-ulnar deviation at the wrist, or of pronation and supination of the forearm. Tremors also occur in the ankles and less frequently in the head and jaw. The tremors increase with emotion and can be controlled to a certain degree by voluntary use of the muscles. They cease during sleep except in advanced cases. It is not uncommon to find that the tremors vary inversely with the degree of muscular rigidity. There may also be cramps in the calf with plantar flexion of the toes, the big toe being hyperextended. Injection of 1% Novocain into the motor point of a muscle will abolish the rigidity, but the tremors persist. The voice is monotonous and it may be high pitched. If the patient is watched when he is sitting it may be noticed

that he remains quite still, apart from the tremors, for several hours. This is known as "poverty of movement." Trophic changes may be seen in the skin of the hands and feet, which appears smooth and glossy. There may be marked sweating, at times unilateral. There is no evidence of a pyramidal tract lesion, thus the deep reflexes are not increased and the plantar response is flexor. Further the posterior columns of the cord and the higher sensory paths are intact, as shown by the absence of sensory changes. In the later stages the patient becomes bedridden and salivation may be troublesome.

**Differential Diagnosis.** There is little difficulty in recognising a typical case. At times the tremor is very slight (paralysis agitans sine tremore), but there is then definite rigidity and the face is usually expressionless. The aching in the legs may be mistaken for intermittent claudication. In senile tremors, the musculature is not rigid, and the expression is normal. The tremors have not the rhythmicity of paralysis agitans. The Parkinsonian syndrome following encephalitis lethargica (see p. 324) can be differentiated by the history of a febrile illness some time previously. This may, however, have been of a slight nature, and have passed almost unnoticed. The age incidence, too, is usually much lower, and the rigidity is more marked than are the tremors. Salivation is common in post-encephalitic Parkinsonism, and oculogyric crises (see p. 324), fits and disturbances of sleep may occur. A Parkinsonian syndrome may also result from syphilis, and poisoning with barbitone, manganese, carbon monoxide and carbon dioxide, or from cerebral arteriosclerosis in old people. In the latter variety the trunk and legs are chiefly affected and tremors are slight.

**Course and Complications.** The course is slowly progressive, all the voluntary muscles become unduly rigid, and the tremors tend to spread from limb to limb, the patient ultimately becomes bed-ridden, and is aptly described as a living statue, who cannot speak, read or write, but whose intelligence remains clear. Complications, such as bronchitis or bronchopneumonia, may develop, or a gradual failure of mental function may set in with a terminal coma.

**Prognosis.** The patient may live for 10 years or more after the onset of symptoms.

**Treatment.** The patient should be kept warm and in as good a general condition as possible. Hyoscine is the most satisfactory drug to alleviate the tremors. It may be given by mouth as a tablet of hyoscine hydrobromide gr. 1/200 to gr. 1/50, 2 or 3 times a day, for several years. The muscles may be lightly massaged and passive movements employed to relieve rigidity. Aching is assuaged by aspirin gr. 10 as required. Sleep is secured by sod. brom. gr. 20, or Trional (methylsulphonal B.P.) gr. 10, or Veronal (barbitonum B.P.) gr. 5, at night. In the terminal stages good nursing is essential.

### Progressive Lenticular Degeneration

(*Hepato-lenticular Degeneration. Wilson's Disease*)

**Definition.** A progressive disease characterised by rhythmic tremors, muscular rigidity and cirrhosis of the liver.

**Etiology** Hepato lenticular degeneration may be due to an unknown toxin *Predisposing causes* 1 Age Children and young adults The average age is 15 years 2 Familial incidence There is a tendency for the disease to occur in more than one member of a family

**Pathology** Bilateral degeneration occurs in the lenticular nucleus, especially in the putamen in which cavities may form The extra-pyramidal motor tract, the lenticulo rubro-spinal tract is presumably deranged Multilobular cirrhosis is seen in the liver The pseudo-scleroses of Westphal-Strumpell, and the torsion spasms of Schälbe Ziehen are closely allied conditions

**Clinical Findings** In some cases a history suggestive of a previous disturbance of the liver is obtained, such as jaundice, with fever and vomiting

**On Examination** Muscular rigidity is found in the limbs and trunk, and bilateral tremors with involuntary movements are seen in the limbs The tremors are increased by voluntary actions, but cease during sleep The face may have an emotional expression, such as a spastic smile The patient laughs or cries without adequate reason Greenish brown pigmentation may be seen at the edge of the cornea in some cases There is general muscular weakness and difficulty in balancing Later flexion contractures occur in the arms and legs, with generalised body wasting In the terminal stages the legs may be extended, and both speech and swallowing are disturbed There is no alteration in the cutaneous sensation and the deep reflexes are normal The liver is not usually palpable

**Differential Diagnosis** The age incidence serves to distinguish the disease from paralysis agitans, and the facial expression also differentiates it from paralysis agitans or the Parkinsonian sequelæ of encephalitis lethargica.

**Course and Complications** The course is variable, being either acute, subacute or chronic, the patient finally becoming bedridden

**Prognosis** The disease is fatal Death occurs in a few weeks to a few years

**Treatment.** There is no cure known

## Migraine

### (Sick Headache)

**Definition** Paroxysmal headache, usually hemicranial

**Etiology** The cause is not known *Exciting causes* 1 Worry 2 Mental or physical strain 3 Eyestrain 4 Indigestion 5 Menstruation *Predisposing causes* 1 Age The attacks usually begin in childhood, and recur until after middle age 2 Sex Females predominate 3 Intellectual ability This is often above the average 4 Heredity In some instances migraine runs in families 5 Other illnesses Migraine may be associated with malaria or gout There is no evidence that migraine is associated with disturbance of the gall bladder

**Pathology.** Various theories have been proposed to account for migraine. They include: 1. Cerebral changes, such as spasm or dilatation of the vessels, or oedema of the occipital cortex. 2. A circulating toxin. 3. Eyestrain. 4. Anaphylaxis. 5. Intermittent hydrocephalus of one lateral ventricle due to temporary obstruction of the foramen of Monro. 6. Pituitary disturbance. 7. A neurosis. There are points in favour of each of these theories, but none is entirely satisfactory. The discovery that in women an attack of migraine is nearly always preceded by the presence of prolan in the urine, suggests there is a disturbance of pituitary function. Normally,  $\alpha$ -strone is present in the urine of women during their menstrual life, but during pregnancy prolan or an anterior pituitary-like body is found in the urine (see p. 667).

**Clinical Findings.** The patient often knows on waking that an attack will develop during the day. He has a feeling of malaise, with slight headache, mental dulness, depression, yawning, cold sensations or giddiness. In the course of a few hours, further premonitory symptoms may occur, such as disturbance of vision. The central vision may be blurred or a portion of the visual field cut out. There may be a definite hemianopia. A bright spot may appear on the dark visual field, or there may be flashes of light or irregular objects with coloured outlines are seen, called fortification spectra (teichopsia). In some cases there is a sensory aura, with a feeling of numbness or tingling gradually travelling from the hand up the arm, usually on the side opposite to that on which the headache develops. It may spread to the lips and tongue on both sides. The headache gradually develops, often on the side opposite to that of the affected visual field. The pain, of a boring nature, often starts in the outer part of one eyeball and spreads over one side of the head and to the upper jaw, in the region of the molar teeth. It may be definitely throbbing in character, and almost intolerable when the patient is standing, but relieved by lying. It is aggravated by movement, noises or bright lights. Both sides of the head may be affected. The patient often feels sick, and may finally vomit, with relief to his pain. In some cases there is a temporary disturbance of speech, such as the use of wrong words.

**On Examination:** The patient usually looks ill and pale, but at times the face is flushed. The temperature is normal. The blood may show an eosinophilia of about 5 to 15%. Ophthalmoplegic migraine occurs in some cases, with paralysis of the III, IV or VI nerves on one side. The headache in these cases is usually very intense. This may last for a few days or weeks. It is possibly due to unilateral hydrocephalus. In other cases a patient who is subject to migraine may experience weakness in one limb or one half of the body on waking, which gradually passes off during the day, without any headache developing.

**Differential Diagnosis.** The nature of the headache is usually diagnosed by the hemicrania and periodical recurrence. An occipital tumour may give rise to a clinical picture almost indistinguishable from migraine. In epilepsy the sensory aura is of much shorter duration than that met with in migraine.

**Course and Complications** The attacks usually last for a day and the patient does not feel quite normal for another 2 or 3 days. They may recur once or twice a week or only at intervals of months. Migraine may be followed later in life by epilepsy, or a migrainous subject may beget an epileptic.

**Prognosis.** The attacks usually cease after the age of 50.

**Treatment** In mild cases a dose of aspirin gr 5 to 20 will usually abort the attack. In more severe instances work is impossible, and the patient has to lie down in a dark and quiet room. Some patients obtain relief from phenacetin gr 10 and caffeine citrate gr 5 repeated in an hour if necessary. It is better to avoid the regular use of bromides, but if the attacks are very frequent and disabling the use of Luminal (phenobarbitonum B.P.) gr  $\frac{1}{2}$  to 1 every night may hold them in control. Ergotamine tartrate will in the majority of cases stop the headache quickly. It is given as a subcutaneous injection or by mouth. Femergin is put up in 1 ml ampoules containing mg 0.5 of ergotamine tartrate or as tablets containing mg 1. A test dose of 0.5 ml can be given subcutaneously repeated in 2 hours if the headache persists or a mg 1 tablet may be taken by mouth and repeated in an hour. If nausea or vomiting is provoked by the ergotamine it can be relieved by a subcutaneous injection of atropin sulph gr 1/100. Ergotamine has no effect upon the frequency of the attacks. The patient must lie down for an hour after the administration of Femergin. Nitroglycerin has a reputation for diminishing the frequency of the attacks and may be given as the tabella glyceryl trinitrat gr 1/1.0 t.d.s. or as Gowers nuxture. Liq trinitrin m 1 liq strychnin m 5 sod brom gr 10 acid hydrochlor dil m 10 tnc gelsem m 5 q chlorof ad fl oz  $\frac{1}{2}$  fl oz  $\frac{1}{2}$  ex aqua t.d.s. Between the attacks the patient should sedulously avoid constipation and any ocular refractive error or muscular imbalance should be corrected.

### The Tics

#### (Habit Spasms)

**Definition** Repetitive purposive movements, originally performed in response to a mental or physical stimulus which tend to persist after the exciting cause has been removed.

**Etiology** In a few cases an external physical cause has existed such as conjunctivitis or a frayed collar. In the majority of instances however no such cause can be traced. There is a neuropathic tendency which leads to the perpetuation of the tic. *Predisposing causes* 1 Age. The tic often starts about puberty, but it may begin earlier or later. 2 Sex. The incidence is equal.

**Pathology** No organic lesion of the nervous system can be found. A tic may be regarded as a conditioned reflex the stimulus being an associated psychical one.

**Clinical Findings** Certain varieties of tic are described, such as 1 *Simple tic*. Here only individual muscles or a few muscles are involved. Thus the patient blinks frequently when talking or twitches his mouth or eyebrow. He may constantly turn his head, shrug his

shoulder, cough or sniff. The same tic is repeated at short intervals, and is likely to be more pronounced if the patient is nervous.

2. *Co-ordinated and convulsive tics.* The movements are more complicated, thus the patient may repeatedly make stooping movements as he walks along, or he may have attacks of convulsive movements accompanied by explosive words, such as swearing (coprolalia), or words or actions may be repeated or copied (echolalia and echokinesis).

3. *Psychical tics.* The patient is the victim of various obsessions. Thus when walking he feels he must touch each lamp-post he passes, and if he misses one, he goes back and touches it.

**Differential Diagnosis.** The brisk nature of the repetitive muscular contractions usually renders the diagnosis of a tic clear. In some cases the movements which occur in chorea, focal epilepsy, torsion spasm (see p. 342), or in encephalitis lethargica, may require consideration.

**Course and Complications.** Tics usually continue without change. A psychical tic may prove so distressing as ultimately to lead to insanity or suicide.

**Prognosis.** The chance of a tic disappearing is less if it has persisted for a long time or if it begins after middle age.

**Treatment.** Any reflex source of irritation should be removed. The general health should be improved as far as possible by proper exercise, and a sufficiency of fresh air and food. Parents should not direct attention to the tic, as, although this may lead to the disappearance of one tic, it is usually attended by the development of a fresh one. An endeavour should be made 2 or 3 times a day to relax the muscles affected, the patient lying down and concentrating his attention on keeping the muscles still. Further, exercises should be performed in which the affected muscles are contracted and relaxed systematically. In some cases hypnotic suggestion has been attended with success.

### Professional Cramp

**Definition.** Fatigue and cramp affecting groups of muscles employed in skilled occupations.

**Etiology.** The cramp occurs as the result of prolonged use of certain muscles, associated with a neuropathic tendency.

**Pathology.** The fatigue process is believed to occur, not in the muscles or in the peripheral nerves, but centrally in the brain.

**Clinical Findings.** Many varieties are described, such as writer's cramp, musician's, telegraphist's, typist's, haircutter's, and cigarette-maker's cramp. The patient is usually a male between the ages of 20 and 50, and a skilled worker who is performing his specialised task. In the early stages there is aching or stiffness in the muscles, and if the worker persists, he may have to stop owing either to definite weakness or cramp of the muscles. After a short rest and rubbing the muscles, he is able to resume. If, however, he endeavours to persist in his occupation, the disability becomes more marked, and finally it is impossible for him to carry on.

**Differential Diagnosis.** Every case should be carefully examined to exclude such lesions as *peripheral neuritis, tenosynovitis, or an early*



stage of paralysis agitans. The diagnosis is usually clear, as certain groups of muscles are affected, and only when a definite act is performed.

**Course and Complications.** The course is progressive, as described above.

**Prognosis.** The outlook is unfavourable unless adequate treatment is given, and even then relapse is liable to occur.

**Treatment.** A rest of at least 6 months from the provocative occupation is required. In writer's cramp the patient should learn to use a large penholder, he should not grip it tightly, and he should write from the shoulder rather than from the fingers and wrists.

## Hysteria

### (*Pathiatism Suggestion Neurosis*)

**Definition.** A condition characterised by signs and symptoms which have resulted from suggestion and which are curable by psychotherapy.

**Etiology.** The true cause of hysteria remains unknown. It is believed by some that there is a restriction of the fields of consciousness, and that the symptoms develop so that the patient may escape from the realities of mental emotions or physical hardships. They result, therefore, either from auto- or hetero-suggestion. The Freudian school consider that hysteria is due to a repression of conflicts, usually of a sexual nature. Various theories have been propounded to account for hysteria. The Greeks believed that the uterus was transplanted to different positions in the body during an attack. Charcot pointed out that hysteria occurs in men as well as in women, and that certain characteristic stigmata are demonstrable between the attacks. Babinski believed that the stigmata result from suggestion on the part of the doctor. Janet introduced the view mentioned above, that hysteria is due to a restriction of the field of consciousness. Hysteria may follow a severe mental or physical shock, or be due to suggestion, especially in epidemics of hysteria. *Predisposing causes.* 1 Age. Usually between 15 and 35 years. 2 Sex. Females predominate. 3. Heredity. A neuropathic tendency is an important factor. 4 Race. In Europe the Teutonic races are comparatively immune, the Latins, Slavs and Jews being susceptible. 5 Class. Hysteria is more common in the hospital class of patient. In the 1914-18 war it was more prevalent amongst privates than amongst officers.

**Pathology.** No organic lesion is found in the brain. There is believed to be a functional disturbance, possibly of the cerebral cortex.

**Clinical Findings.** The patient is usually an adult woman, lacking emotional control and introspective. She is not worried about her symptoms, and may be mentally dull. The clinical picture varies according to the type of hysteria present.

1. *Motor Types.* (a) *Convulsive hysteria.* This occurs in attacks, and is sometimes known as hysterical epilepsy. *Minor attacks.* The patient may have preliminary disturbances such as palpitations, the

feeling of a lump rising in the throat (*globus hystericus*) or distressed breathing. She then falls in the presence of onlookers, but is careful not to injure herself. Various clonic muscular contractions take place, but the patient is not really unconscious, she does not bite her tongue, or micturate, and the corneal reflexes persist. *Major attacks (grande hystérie)*: This is very uncommon in England, but was met with chiefly in France at the end of the last century. The attack may last for several hours. The early stages are characterised by clonic and tonic muscular contractions. The patient may scream and assume various contortional and emotional attitudes. On recovery, visual and auditory hallucinations may persist for a time. The attacks occur in the presence of others, and probably are largely due to suggestion. Somnambulism is considered to be a manifestation of major hysteria. (b) *Non-convulsive motor hysteria*. The patient may complain of inability to move an arm or leg, or the arm and leg on the same side of the body, or both legs.

*On Examination*: Various types of motor hysteria may be seen, such as a monoplegia, hemiplegia or paraplegia. Further, there may be contractures, irregular movements or catalepsy. In the hysterical paralysis certain distinguishing features can usually be detected. Thus a patient may say she cannot stand or walk (*astasia abasia*) and yet she can move her legs perfectly when lying down. The muscles do not waste, the electrical reactions are normal, and the deep reflexes are often exaggerated. The plantar response is flexor in type. If a patient is asked to bend a paralysed arm, the antagonistic muscles can often be felt to contract, opposing any flexion result which would be produced by contraction of the agonists. In a hysterical paralysis of a leg, if the patient is asked to raise her body when lying with her arms across her chest, the sound leg will come up in the air, but the paralysed one remain on the bed. This is due to muscular contraction in the paralysed leg, and is the reverse of that which takes place in an organic monoplegia, where the paralysed leg rises higher than the sound one. Further, in hysterical monoplegia, sensory cutaneous loss of a hysterical type (see below, p. 348), is generally present. The gait differs from that of an organic lesion, in that the "paralysed" leg tends to be drawn along after the body, and is not swung outwards as in an organic lesion. The adductor muscles of the vocal cords may be affected (see p. 120). *Spasmodic contractions*. Various muscles of the trunk, limbs or face, may be involved. Thus there may be trismus, or ptosis due to contraction of the orbicularis palpebrarum, without compensatory wrinkling of the forehead. The contraction does not usually cease during sleep, but is abolished by anaesthesia. The phantom abdominal tumour is of this nature, there being spasms of the abdominal muscles in localised areas, with some flatulent distension. *Irregular movements*. These consist of tremors, muscular twitchings, and choreiform or repetitive movements. Various complicated actions, such as bowing, may be repeatedly performed. There may be attacks of laughing or crying. *In catalepsy* the patient goes into a trance, the limbs are rigid, and remain in whatever position they are placed.

2. *Sensory Types*. The patient may complain of every imaginable

type of pain. There may be headache, like a nail being driven into the skull (*clavus hystericus*), defective vision, such as mistiness, inability to bear a bright light, or blindness in one eye. The patient may complain of deafness or increased sensitivity to sounds. Other symptoms include deficiency of taste (often bilateral) or of smell, a lump in the throat, loss of appetite, flatulent dyspepsia, constipation, retention of urine, yawning, hiccough, coughing, inability to take a full breath, palpitations, sweating, or flushing. *Psychical types* include cases of dual personality and amnesia. The patient is reported as "nussing and probably suffering from loss of memory."

*On Examination.* In hysterical blindness of one eye, the patient may be seen to blink when a blow is directed at the affected eye, the other being covered. In hysterical deafness, the patient may be awakened by a noise of average intensity. Certain hysterical stigmata may be found, the most important being cutaneous and pharyngeal anæsthesia, and restriction of the visual fields. The patient is unaware of these stigmata, and some authorities maintain they result from suggestion on the part of the examiner. This is doubtful, they are more probably due to auto-suggestion. *Cutaneous anæsthesia.* The patient does not injure the anæsthetic part. There may be hemianæsthesia, or a sock, stocking, glove or sleeve anæsthesia in a limb. The upper limits of anæsthesia are fairly sharply defined. Joint sense is unaffected. The patient may really feel when the skin is stimulated, as is shown by asking her to say "yes" when the skin is pricked, and "no" when it is not pricked. She may fall into the trap and say "no" when the pin is applied to the anæsthetic area. A stimulus applied to one limb may be referred to a corresponding spot on the other limb (*allocheiria*). Areas of hyperæsthesia may be found on the chest or abdomen, and so-called hysterogenous spots. These are tender spots, especially likely to be present in the left inguinal region. Pressure on them may induce a hysterical fit. *Reflexes.* The conjunctival, abdominal and plantar reflexes may be lost in cases of cutaneous anæsthesia. The pharyngeal reflex is not necessarily lost in hysteria if care is taken to avoid suggestion. *Restriction of the visual fields.* The patient does not complain of loss of vision in any direction before perimetric tests are carried out. The spiral restriction of the visual field, as determined by the perimeter, appears to be the result of suggestion.

*Differential Diagnosis.* Convulsive hysteria is differentiated from epilepsy by the points mentioned on p. 332. In many cases the diagnosis of hysteria presents no difficulties, when the characteristic findings described above are considered. It should always be remembered that a hysterical element may be superimposed on an organic lesion, and may persist when the organic lesion is healed or arrested. In every case a complete examination should be carried out, not once only, but at intervals of several weeks. In this manner the early stages of an organic disease, such as disseminated sclerosis, will not be overlooked. In malin<sub>g</sub> there is a deliberate attempt to deceive, which is not the case in hysteria.

*Course and Complications.* Hysterical symptoms may persist for

many years or disappear suddenly at any time. There is always a tendency for the cure to be temporary and for subsequent recurrence.

**Prognosis.** "Miraculous" cures may result from suggestion, the blind recovering his sight or the mute his power of speech.

**Treatment.** In some cases a convulsive attack may be arrested by firm pressure on a so-called hysterofrenic spot, such as the supra-orbital notch. The most hopeful method of cure is by persuasion. The physician must acquire the complete confidence of his patient, who is made to believe that a cure is possible, and will be effected. The nature of the complaint is explained to the patient. In some cases the use of electrical stimulation is of value in abolishing cutaneous anæsthesia or in demonstrating to the patient that his muscles will contract. If the patient is very excited, the following mixture is of value: Sod. brom. gr. 10, inc. sumbul. (B.P.C.) m. 15, sp. chlorof. m. 7, infus. gent. co. rec. ad fl. oz. 1. Fl. oz. 1 t.d.s. p.c. Hypnotic and psychoanalytic treatment are not recommended.

### Neurasthenia

#### (Psychasthenia. Anxiety States)

**Definition.** A condition characterised by abnormal fatigue of the mind or body without a discoverable organic cause.

**Etiology.** No definite cause can be assigned to neurasthenia; it is sometimes considered to be a fatigue neurosis. Pavlov has produced experimental neurosis in dogs as the result of overtaxing inhibition processes by conditioned reflexes. *Exciting causes:* 1. Overwork. 2. Worry. 3. Illness, such as influenza, anæmia or enteric fever. 4. Drugs, such as alcohol or cocaine. 5. Injury, producing traumatic neurasthenia. *Predisposing causes:* 1. An inherited neuropathic tendency. 2. Age: Usually between 20 and 45 years. 3. Sex: Males predominate.

The Freudian school believes that neurasthenia is due to some sexual trauma, which may only be made manifest when one of the exciting causes is operative.

**Clinical Findings.** The patient is usually an adult of lugubrious expression and with many complaints, although in some cases he looks extremely fit. In order to avoid forgetting any symptom he may bring a list of them written out. The examiner usually finds that the list is not easily exhausted, and when asked if there is anything else that he has noticed wrong, the patient will produce another complaint. In neurasthenia bodily complaints predominate, whereas in psychasthenia the symptoms are chiefly mental. The onset is usually gradual, the patient feeling tired and lacking interest in anything except himself. The fatigue is abnormal, the patient wakes feeling tired, but often improves as the day goes on. The symptoms in neurasthenia may be referred to the various systems of the body. The patient may complain of loss of appetite, flatulent dyspepsia, or of occipital headache. He often states that there is a feeling of oppression on the top of the head or of constriction of the head. The skull may feel empty or too full. He may suffer from palpitations, dizziness, flushing, sweating, a frequent

desire to yawn, a sensation of a lump in the throat, numbness or tingling in various parts of the body. Backache may be marked and sexual disturbances, such as impotence, a prominent symptom. In psychasthenia the patient may have groundless fears. Certain of these have been granted names, such as fear of open spaces (agoraphobia), of closed spaces (claustrophobia), of disease (pathophobia), of crowds (anthropophobia), of solitude (monophobia), and of dirt (mysophobia). The patient feels unable to concentrate, to read or to keep still. He has needless worries and suffers from insomnia. He may be the victim of various obsessions, a prey to drink (dypsomania), to drugs (toxicomania), or to stealing (kleptomania).

**On Examination** The patient often looks anxious, but the nutrition is usually good. The peripheral circulation may be poor and the hands cold and bluish. There are no signs of organic disease discoverable, but the deep reflexes are brisk. In the cases of flatulent dyspepsia the patient may make loud eructations the whole time that he is being examined. The blood pressure in neurasthenia is usually low, and the pulse rate unstable, varying more than 10 beats in the standing and recumbent position. Traumatic neurasthenia follows some accidental injury, such as a railway accident when often no external injury is visible. Railway spine may then develop, with pain in the back and weakness, although no organic lesion is discoverable by X ray or other examination.

**Differential Diagnosis** Every care should be taken to exclude organic disease before a diagnosis of neurasthenia is made. Thus early pulmonary tuberculosis is at times diagnosed as neurasthenia, a mistake which would not occur if the morning and evening temperature were taken, and the chest carefully examined physically and radiographically. A cerebral tumour in a silent area of the brain may also cause disturbance of health with vague sensations of distress and pain, without any localising signs being found. Later, with increasing intracranial pressure the diagnosis becomes apparent. General paralysis of the insane at its onset may be mistaken for neurasthenia. If there is doubt, the blood and cerebro spinal fluid should be examined. An early case of Addison's disease may also be diagnosed as neurasthenia, a blood pressure as low as 100 mm Hg systolic should suggest the former diagnosis.

**Course and Complications** Neurasthenia is a chronic condition, which is liable to persist unless adequately treated. Insanity is a rare complication.

**Prognosis** The outlook is fairly good, but, despite treatment, the neuropathic disposition remains and relapses are not infrequent.

**Treatment** The most important factor in successful treatment is the discovery and elimination of the exciting cause, whether it is due to physical strain, mental worry, toxæmia or trauma. In severe cases a complete rest, change of environment, and a congenial occupation are required. Apart from this the treatment is general, such as extra diet in the form of a glass of milk or cup of cocoa in the morning, some exercise daily out of doors, and early hours at night. If there is difficulty

in sleeping, a dose of sod. brom, gr. 15 to 20 should be given at night. A tonic such as syrup. glycerophosph. co. (B.P.C.) is also of value, in doses of m. 60 t.d.s. p.c. Psychoanalytical treatment is not recommended. In traumatic neurasthenia an improvement is often noted when the responsibility for treatment is definitely cast upon the patient, by settlement of any compensation claim.

## THE CEREBELLUM

### Anatomy and Physiology

The cerebellum is composed of three parts, a central vermis and two lateral lobes. The vermis contains the following nuclei: The nucleus fastigii (roof nucleus) and the nucleus globosus on each side. Each lateral lobe contains the dentate nucleus and the nucleus emboliformis. The cerebellum is connected with the brain by three peduncles on each side, the inferior peduncle or restiform body, the middle peduncle or brachium pontis and the superior peduncle or brachium conjunctivum (see Fig. 31, facing p. 356).

**The Efferent Tracts.** There are three main efferent tracts from the cerebellum.

1. *The Cerebello-rubro-spinal Tract.* From the cortex of the lateral lobe through the superior peduncle to the opposite red nucleus. The second relay of fibres conducts the impulses down the rubro-spinal tract which crosses at its origin so that it runs down to the anterior horn cells on the same side of the body as that from which the impulses arose in the cerebellum. The final relay is by the anterior nerve roots to the skeletal muscles.

2. *The Cerebello-cerebral Tract.* From the cerebellar cortex to the dentate nucleus where the second relay passes through the superior cerebellar peduncle to the thalamus of the opposite side. The final relay conducts the impulses to the frontal and post-central cerebral cortex.

3. *The Cerebello-Deiters' Tract.* From the cerebellar cortex through the inferior peduncle to Deiters' nucleus of the same side. Relays of fibres connect thence with the vestibulo-spinal tract running down to the anterior horn cells on the same side, and so by the final relay to the skeletal muscles, and by the medial longitudinal bundle with the III, IV and VI nuclei regulating eye movements and with the nuclei of the spinal portion of the XI nerve controlling head movements.

**The Afferent Tracts.** There are six main tracts.

1. *The Direct Cerebellar or Dorsal Spino-cerebellar Tract of Flechsig.* This runs through the inferior peduncle to the vermis on the same side. The final relay carries the impulses to the cerebellar cortex of the lateral lobe of the same side.

2. *The Indirect Cerebellar Tract or Ventral Spino-cerebellar Tract of Gowers.* This runs up the opposite side of the cord, and ascends to the level of the red nucleus. It turns down and crosses the mid-line to the superior cerebellar peduncle, and so passes to the vermis. The final relay carries the impulses to the cerebellar cortex of the lateral lobe on the side at which the impulses entered the spinal cord.

3 *Fibres arising from the Cuneate and Gracile Nuclei* These form the superficial and deep arcuate fibres, and pass through the inferior peduncle to the vermis. The final relay is to the cortex of the lateral lobe of the cerebellum, on the same side as that on which the majority of impulses entered the cord.

4 *The Olivary cerebellar Tract* Fibres pass from the inferior olive in the medulla cross the mid line, and enter the cerebellum by the inferior peduncle. They relay in the vermis, the final path being to the cerebellar cortex of the lateral lobe on the opposite side.

5 *The Deitero cerebellar Tract* Fibres run from Deiters' nucleus through the inferior peduncle to the homolateral nucleus fastigi of the vermis. A second relay conveys the impulses to the cerebellar cortex of the lateral lobe of the same side.

6 *The Cerebro cerebellar Tract* Fibres pass from the frontal, temporal and occipital lobes to the pons. A second relay conveys the impulses by the middle peduncle to the vermis. The final relay runs to the cerebellar cortex of the opposite lobe.

The cerebellum is thus intimately connected with the skeletal muscles on both sides with the impulses from Deiters' nucleus concerned with equilibrium and with the co ordination of the eye and head movements.

The chief function of the cerebellum is to enforce and control the postural tone of the skeletal muscles which is of vital importance for synergic or co ordinated movements. The hemispheres are chiefly concerned with the tone of muscles on the same side of the body, and the vermis with the backward or forward balance of the body and with the conjugate deviation of the head and eyes, phonation and articulation.

### Lesions of the Cerebellum

The following lesions may occur. 1 *Hypoplasia* This is a congenital lesion. The cerebellum may be partly or completely absent.

2 *Primary Progressive Degeneration* (Singer Brown ataxia, see p 140) The fibres running from the cortex to the nuclei are affected.

3 *Oligo ponto cerebellar Atrophy* There is atrophy of the cortex, middle peduncles and part of the inferior peduncles. The inferior olives are also affected.

4 *Vascular Lesions* Thrombosis is liable to affect the posterior inferior cerebellar artery. Hæmorrhage may occur from the superior cerebellar artery.

5 *Tumours* These include a glioma, endothelioma, sarcoma, psammoma lipoma myxoma and cholesteatoma. Degenerative cysts also occur, they are generally gliomatous or sarcomatous.

6 *Granulomata* A tuberculoma or gumma may be found.

7 *Abscess* This may be secondary to suppuration in the mastoid or labyrinth or to spread of infection from lateral sinus thrombosis. The infection may be blood borne, as in bronchiectasis or infective endocarditis. In some cases the abscess results from a wound of the cerebellum, which may have been inflicted some time before.

8 *Disseminated Sclerosis*

The most commonly occurring lesions are a glioma and a tuberculoma.

**Clinical Findings.** The results of cerebellar lesions vary according to whether they are irritative or paralytic, and also whether the lateral hemisphere or vermis is affected. Neighbouring structures may also be involved, such as the cranial nerves, especially the VIII, VI and V, and the pons with disturbances of either the motor or sensory tracts.

*Irritative lesions.* These result usually from a wound or hæmorrhage.

*Paralytic lesions.* These may be due to thrombosis, a tumour or an abscess.

### Cerebellar Hæmorrhage

The patient complains of very severe vertigo. He feels as if his body is being rotated away from the side of the lesion, and as if surrounding objects are also rotating in the same direction. There may also be pain in the occipital region with vomiting, and the patient often falls to the ground, his body being rotated on its long axis by "forced movements."

*On Examination:* The patient is often found lying with the side of his face, corresponding with the site of the lesion, on the pillow. Spontaneous nystagmus may be seen, with slow movements away from the side of the lesion and sharp short movements in the opposite direction. There may also be skew deviation of the eyes, the eye on the affected side looking downwards and inwards, and the other eye upwards and outwards. In many cases a hæmorrhage involves the lateral region of the medulla. When the spino-thalamic tract is affected, there is analgesia for pain and temperature sensations on the same side of the face and head and on the opposite side of the body and limbs. Involvement of the nucleus ambiguus may result in interference with swallowing and speech. An irritative lesion may cause a cerebellar fit, characterised by a sudden onset, with loss of consciousness, and tonic spasm. The head is retracted and the back arched. The elbows, hips, knees and ankles are extended. The patient does not usually bite his tongue, or micturate. With unilateral lesions the homolateral leg is adducted and the heterolateral leg abducted. The patient rotates to the side of the lesion, and the eyes deviate to the opposite side.

### The Cerebellar-syndrome

Special signs and symptoms are described which constitute the "cerebellar-syndrome." These signs cannot be detected in every case, and are obscured if the lesion presses on the medulla and interferes with the pyramidal tracts. The chief features of the cerebellar-syndrome are as follows: 1. *Hypotonia.* The muscles of the body on the same side as the lesion are affected. This may be demonstrated when the patient is lying down by the extent to which the patella can be depressed on the two sides when the muscles are relaxed. The leg can also be abducted farther on the affected side. The arm is more flail-like if the forearm is shaken up and down by grasping the upper arm. In the past-pointing test (see p. 379) the arm on the affected side deviates away from the object.



2 *Asthemia* The grip is less powerful on the affected side, and the limb will tire suddenly and completely

3 *Ataxia* This can be shown in various ways Thus there is *asynergia* or decomposition of movements The muscles do not work together smoothly in performing such an action as putting the foot on a chair *Astasia* consists in jerky clonic contractions of muscles and can be tested by feeling the biceps while the patient flexes his forearm with his elbow resting on a table *Dysmetria* (disturbance of the range of movement) Movements are often performed excessively (*hypermetria*) Thus there is a tendency to overshoot the mark in the finger-nose test and in taking up a glass the hand opens too widely when seizing it and when putting it down There may also be delay in relaxation of muscles so that the glass is knocked over when the patient takes his hand away from it Associated movements Irregular clonic movements may occur in the *homolateral limb* when forced muscular contractions are made in the limb on the sound side In some cases there is a tendency on standing to fall backwards or to one side *Cerebellar ataxia* is not increased on closing the eyes *Ataxia of speech* occurs especially in vermis lesions the speech being either staccato or drunken

4 *Gait* This is reeling or drunken the patient tending to deviate or fall to the affected side There is difficulty in standing on the leg on the affected side On walking also the affected leg swings outwards, the patient tending to walk round it as if it were a crutch The arm on the affected side often remains motionless *Attitude* The occiput may be pointing towards the shoulder on the affected side which is usually slightly lower than, and in front of the opposite shoulder, there is some scoliosis with concavity towards the side of the lesion

5 *The Rebound Phenomenon* The patient holds his arms flexed, and the examiner pulls against them On releasing the pull on the arm on the affected side the patient's hand flies back to hit his face or body whereas this movement is automatically checked on the sound side

6 *Dysidiadochokinesis* The patient is unable to perform rapid alternate actions on the affected side such as pronation and supination of the forearm or closing and unclosing the hand

7 *Nystagmus* This occurs typically as a fixation nystagmus which is not present when the eyes are at rest If the patient is asked to look to the affected side nystagmus appears the eyes slowly swinging away from the fixation position owing to lack of tone in the muscles, and being rapidly jerked back again If the patient looks to the sound side the nystagmus consists of smaller movements which are more rapid and irregular but there is a slow movement away from the fixation position and a rapid jerk back With bilateral lesions nystagmus occurs on looking to either side the slow movement being away from the fixation point and the quick movement towards it Vertical nystagmus may occur with lesions of the vermis Nystagmus may also be provoked by caloric stimulation of the ears Further, if the stimulation produces conjugate deviation of the eyes, the lesion is also probably

central. There is also often difficulty in looking towards the side of the lesion.

8. *Vertigo*. This may persist, the patient feeling that both he and the surrounding objects are rotating away from the side of the lesion.

9. *The Knee-jerks*. The knee on the side of the lesion shows a pendulum swing due to lack of tone in the extensors.

10. *Tremors*. Intention tremor may occur in the homolateral limbs. If the arms are held out static tremors may be seen on the affected side, the hand dropping a little and then being jerked upwards.

11. *Cerebellar Catalepsy*. This term is applied to the abnormal steadiness which may at first be seen in the homolateral arm or leg when it is held out, before the static tremor develops.

### Cerebellar Thrombosis

**Clinical Findings.** Thrombosis of the posterior inferior cerebellar artery is known as the Wallenberg syndrome. There is usually a comparatively sudden onset with ataxia on the affected side, but the patient does not lose consciousness. In addition, owing to involvement of the medulla, there may be dissociated anæsthesia (loss of pain and temperature sensation, but not of touch) of the trunk and limbs on the opposite side, with homolateral dissociated anæsthesia of the face, and paralysis of the palate and vocal cord. Heparin, mg. 150 (3 mls of a 5% solution), may be injected intravenously t.i.d. for 4 or 5 days with good results.

### Cerebellar Tumours

**Clinical Findings.** The patient is often a child or young adult, who complains of headache, and later disturbance of vision, vomiting and vertigo may occur.

**On Examination :** The generalised increased cranial pressure accounts for the headache, vomiting and optic neuritis. The localised cerebellar lesion gives rise to a variable number of the signs described above as constituting the cerebellar syndrome. Extension of the tumour may cause paralysis of the VI nerve, or, by pressure on the pons, hemiplegia on the opposite side. If a tumour is situated in the vermis, there is a tendency for the patient to fall backwards or forwards.

### Extra-cerebellar Tumours

#### (Ponto-cerebellar Angle Tumours)

**Pathology.** A meningioma arises from the meninges covering the under surface of the cerebellum, and a neurofibroma from the sheath of a cranial nerve, generally the vestibular part of the VIII nerve.

**Clinical Findings.** The patient is usually an adult, who first complains of deafness and tinnitus in one ear. Later there is headache and vertigo, the patient feeling that he is rotating to the side of the lesion, but that objects are rotating away from the side of the lesion.

**On Examination :** Nerve deafness is found on the affected side, and there may be signs of involvement of the VI or VII nerves, or the

sensory part of the V nerve. Diminution or loss of the corneal reflex may be an early sign. The pyramidal tract on the opposite side may be affected by pressure of the medulla against the skull. There is then a spastic hemiplegia on the same side as the tumour. Symptoms of a lateral cerebellar lesion may also appear, but these will be largely masked if the pyramidal tract is involved.

### Cerebellar Abscess

**Clinical Findings.** The patient complains of headache, occipital or frontal and there may be vertigo and stiffness of the neck and some drowsiness. A history is sometimes given of recent mastoid or labyrinthine trouble with deafness or tinnitus.

**On Examination.** The patient may be apathetic, the temperature is often raised although sometimes it is normal. Spontaneous nystagmus may be seen, the short quick movement being towards the side of the abscess. The patient may lie on the sound side with the knees flexed. The finger-nose test often shows deviation of the arm on the affected side in the direction of the lesion. Optic neuritis is frequently marked. Muscle hypotonus may be detected on the affected side and dysidiadochokinesis may be easily recognised in the arms.

**Differential Diagnosis.** A cerebellar abscess is often hard to diagnose and may be overlooked. It may give rise to no localising signs. If it closely follows a purulent labyrinthitis no fresh signs or symptoms may be noted. Lumbar puncture serves to differentiate septic meningitis. If nystagmus persists after operation for purulent labyrinthitis there is probably also a cerebellar abscess.

**Course and Complications.** The course is usually progressive. Pyæmia and purulent meningitis may occur as complications.

**Prognosis.** Death usually occurs in from 1 to 2 months unless the abscess can be satisfactorily drained.

**Treatment.** This is surgical, as soon as the abscess has been diagnosed it should be drained.

## THE CRANIAL NERVES

### I The Olfactory Nerve

**Anatomy.** The olfactory nerves receive impulses of smell from the back of the nose. The fibres pass through the cribriform plate of the ethmoid bone and travel back to the olfactory bulb and olfactory tract, which lies in a groove on the orbital surface of the frontal lobe. The olfactory tract divides centrally into two roots, the inner one crosses to end in the uncinate gyrus of the opposite side, and the outer connects with the uncinate gyrus of the same side of the brain.

**Lesions of the Olfactory Nerve.** The nerve endings may be affected in acute or chronic rhinitis. The nerve fibres may be damaged by a fracture of the base of the skull by pressure of a tumour of the frontal lobe or pituitary region, or by basal meningitis. Atrophy of the nerve may occur in *tuberculosis dorsalis*. Toxins such as alcohol and nicotine, and infections such as influenza, may result in loss of smell.

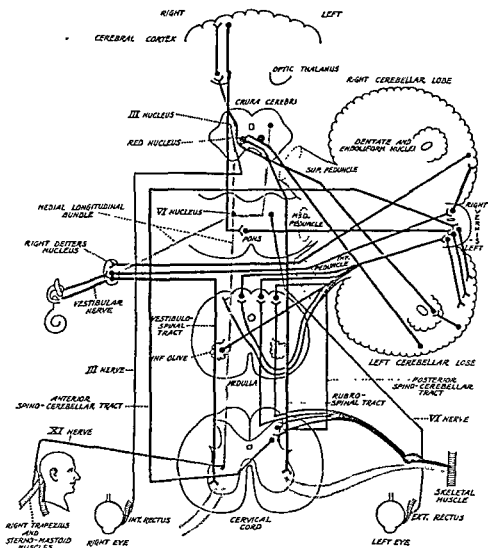


FIG. 31. DIAGRAM OF THE CEREBELLAR CONNECTIONS.

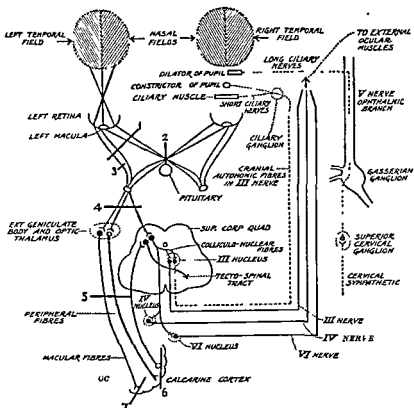


FIG 32. DIAGRAM OF THE VISUAL PATH

*Lesion at 1 (optic nerve) = Blindness of eye*

*Lesion at 2 (centre optic chiasma) = Bitemporal hemianopia.*

*Lesion at 3 (outer side chiasma) = Nasal hemianopia*

*Lesion at 4 (optic tract) = Bilateral homonymous hemianopia, with abolition of light pupil reflex.*

*Lesion at 5 (optic radiation) = Bilateral homonymous hemianopia, with light pupil reflex normal*

*Lesion at 6 (calcarine cortex) = Bilateral hemianopia (peripheral vision).*

*Lesion at 7 (occipital pole) = Loss of central vision, left halves of macula.*

**Clinical Findings.** The sensation of smell is tested in each nostril by asking the patient to inhale such substances as oil of cloves, peppermint, lemon, or asafoetida. Anosmia implies loss of smell, and in such cases food appears tasteless. In hyperosmia the patient is unduly sensitive to smells. This may be a manifestation of hysteria or result from drugs, such as strychnine or cocaine. Parosmia is a state of perverted sensation of smell, a pleasant odour being considered repugnant or *vice versa*. It occurs at times in association with a tumour of the hippocampal gyrus, or with empyema of the antrum. In epilepsy an olfactory aura may precede an attack.

## II. The Optic Nerve

**Anatomy** (see Fig. 32). There are two visual fields, one for each eye, and each may be divided into an inner or nasal, and an outer or temporal part. The retina of each eye may also be divided into two parts, the inner which receives impressions of objects in the temporal field of vision, and the outer, which is the receptor for the nasal visual field. The optic nerve passes from the retina, and meeting its fellow in the mid-line, intermingles to form the optic chiasma, from which the optic tracts proceed back to connect with the primary visual centres. The fibres from the nasal halves of the retinae decussate in the optic chiasma and pass to the primary visual centres on the opposite side of the brain, whereas the fibres from the temporal halves of the retinae are carried on in the homolateral optic tract. The macular fibres from each retina undergo a partial decussation, some fibres crossing in the chiasma and others passing back in the optic tract of the same side. The primary visual centres are the external geniculate body, the superior corpus quadrigeminum and the pulvinar of the optic thalamus. It is very doubtful, however, whether the optic thalamus is a visual centre. From the external geniculate body relays of fibres convey the visual impulses to the cerebral cortex of the occipital lobe, especially to those convolutions situated around the calcarine fissure in the "area striata," on the mesial surface of the occipital lobe. The fibres first pass through the posterior limb of the internal capsule, and then run back in the optic radiation to the occipital cortex. Owing to the crossing of the macular fibres in the optic chiasma, the right half of each macula has its cortical centre in the right occipital lobe close to the occipital pole and the left half of the macula in the left occipital lobe. Fibres also run from the occipital lobe to the optic thalamus and the superior corpus quadrigeminum. The first relay of fibres in the optic tract which ends in the superior corpus quadrigeminum is situated close to the nucleus of origin of the III cranial nerve on the opposite side, and a reflex arc for light impulses is thus established between the retina and the ocular muscles. Connections are also established with the IV and VI nuclei. The III nerve, in addition to conveying fibres supplying certain extrinsic eye muscles, is also the channel for impulses passing to the intrinsic ocular muscles. Thus the cranial autonomic fibres run in the III nerve to the ciliary ganglion, where a fresh relay starts, the short ciliary nerves, supplying the ciliary muscle and the constrictor of the pupil. The

dilator muscle of the pupil is supplied by the sympathetic chain which passes to the superior cervical ganglion. A fresh relay conducts the impulses through the Gasserian ganglion of the V nerve, to the ophthalmic branch of this nerve, and the impulses travel along the long ciliary nerves to the dilator muscle. With these anatomical facts clearly established, the effects of lesions at various points in the visual path are easily appreciated (see Fig. 32).

**Lesions of the Optic Nerve and Visual Path** The most important affections of the optic nerve are neuritis and atrophy.

### Optic Neuritis, Papillitis and Papilloedema

**Definition.** These terms are applied to inflammation and swelling of the head of the optic nerve within the globe of the eye.

**Etiology** Optic neuritis is usually bilateral, and may be due to increased intracranial pressure, resulting in increased tension of fluid in the nerve sheath with compression of veins, or it may be a manifestation of neuro retinitis. The most common cause of increased intracranial pressure is a tumour of the cerebrum or cerebellum. Other causes include cerebral abscess, meningitis, especially when due to tuberculosis, and chronic hydrocephalus. Neuro retinitis may be due to nephritis, diabetes mellitus, syphilis or leukaemia. Unilateral optic neuritis results from orbital periostitis, inflammation of the orbital connective tissue, a retro orbital tumour, localised meningitis, or an aneurysm of the ophthalmic or internal carotid artery.

**Clinical Findings** The patient may complain of headache, but the vision may be apparently normal, although definite papilloedema is present. Later attacks of blurred vision or definite impairment of sight occur, especially of central vision so that reading is impossible.

**On Examination** In the early stages the pupils are normal, but later they are dilated and do not react to light. Ophthalmoscopic examination shows the disc red and swollen, with a blurred edge. The retinal veins are dilated and the arteries are small. In papilloedema or choked disc, there is much oedema and swelling of the disc. In addition, in neuro retinitis retinal changes such as patches of exudate and hæmorrhages are seen.

**Prognosis** This depends upon the cause of the neuritis. When due to syphilis, there is usually a good response to treatment.

**Treatment** A decompression operation should be performed in cases due to cerebral tumour in order to endeavour to save the sight before optic atrophy develops.

### Retro-bulbar Neuritis

**Definition** Inflammation of the optic nerve behind the eye.

**Etiology** Retro bulbar neuritis may be due to—1 Toxic causes, especially tobacco and alcohol. 2 Sepsis in the ethmoidal, sphenoidal or frontal sinuses, in the cranial antra and possibly in other sites of the body such as the teeth. 3 Disseminated sclerosis, syphilis affecting the optic nerve, or a hæmorrhage into the sheath of the optic nerve.

4. Metabolic disturbances, such as diabetes mellitus and rheumatism. Exposure to cold is a predisposing cause.

**Clinical Findings.** When retro-bulbar neuritis develops acutely it is usually unilateral. The patient complains of progressive loss of vision, and often there is pain on moving the eye.

**On Examination:** Pressure on the eyeball may cause pain. In the early stage the pupil appears normal, but it may be found that on exposure to a bright light the pupil first contracts but subsequently dilates while still exposed to the light. The disc is normal. Determination of the visual fields usually reveals a central scotoma (blind spot) where either white objects or certain coloured objects are not seen. This is due to a lesion of the papillo-macular bundle, and is called axial neuritis. The peripheral visual field may alone be restricted in interstitial peripheral neuritis. In more advanced cases sight is completely lost (diffuse neuritis), and there is optic atrophy with pallor of the optic disc.

**Prognosis.** This is on the whole favourable, but relapses may occur and both eyes may be affected.

**Treatment.** This varies with the cause. The patient should stop smoking and take no alcohol. A shade should be worn over the affected eye. Septic foci should be searched for and removed. Syphilis or diabetes mellitus, if present, must be adequately treated (see pp. 248, 630). If no specific cause is found iodides should be given in increasing doses, with sodium salicylate, such as Pot. iod. gr. 5 to 30, sod. salicyl. gr. 5, sod. bicarb. gr. 10, syr. aurant. m. 20, aquam ad fl. oz. 1. Fl. oz. 1 t.d.s. p.c.

### Primary Optic Atrophy

**Definition.** Atrophy of the optic nerve, not preceded by a stage of optic neuritis.

**Etiology.** The atrophy may be due to; 1. Nervous diseases, such as tabes dorsalis, general paralysis of the insane, amaurotic family idiocy, a frontal lobe tumour, and possibly disseminated sclerosis. 2. Toxic substances, such as lead, arsenic in the form of Atoxyl or tryparamide, or methylated spirit. 3. A hereditary variety, known as Leber's disease.

**Clinical Findings.** The patient complains of progressive loss of sight. One or both eyes may be affected.

**On Examination:** The pupil is dilated and does not react to light. The disc is white with a sharp margin, the surrounding retina being normal. The field of vision diminishes from the periphery, without a central scotoma.

**Prognosis.** This is unfavourable.

**Treatment.** This is unsatisfactory. If the optic atrophy is due to tabes dorsalis or general paralysis of the insane, the treatment detailed on pp. 391, 393 should be given.

### Secondary Optic Atrophy

**Definition.** Degeneration of the optic nerve subsequent to optic neuritis.



**Etiology** The causes are similar to those mentioned on p 358 for optic neuritis such as a cerebral tumour, aneurysm or hydrocephalus

**Clinical Findings** The patient complains of loss of sight

**On Examination** The disc is pale but the edges are blurred and the retinal arteries thread like

**Prognosis** This is very unfavourable

**Treatment** No treatment is likely to be efficacious when the stage of atrophy has been reached

### The Optic Chiasma, Optic Tract, Optic Radiations and Calcarine Region

Lesions affecting these portions of the visual path are usually due to tumours vascular disturbances, meningitis or aneurysm of the internal carotid artery

**Clinical Findings** The fields of vision are mapped out with the aid of a perimeter. Normally the visual field is most extensive for white objects the fields for blue, red and green objects being smaller in the order of colours given. The visual fields may be found cut off in definite directions. Thus in bitemporal hemianopia both temporal fields are blind. This results from a lesion affecting the central part of the chiasma (see Fig 32). In the early stages the upper temporal quadrant of one visual field is usually first affected by tumours of the pituitary, which cause pressure from below upwards whereas in suprasellar tumours with pressure from above downwards the early loss is likely to occur in the lower temporal quadrant. In nasal hemianopia the nasal visual fields are eliminated. This is a rare condition due to a lesion affecting the outer part of the chiasma on both sides. It may be due to atheroma of the internal carotid arteries to distension of the third ventricle due to an intracranial tumour or to chronic internal hydrocephalus causing pressure of the outer part of the chiasma against the internal carotid arteries. In bilateral homonymous hemianopia the nasal field of one eye and the temporal field of the other are affected. This results from a lesion of the optic tract or radiation on one side. A pituitary tumour may cause such a lesion. In quadrant hemianopia, one quadrant of the visual field is affected such as the upper half of one temporal and the upper half of the other nasal field or the lower half of one temporal and the lower half of the other nasal field. This results from a lesion around the calcarine fissure the superior quadrant of the visual field being represented below the calcarine fissure, and the inferior quadrant of the visual field above the calcarine fissure. When the occipital pole is affected there may be central blindness half the macula on each side being represented on each occipital pole (see Fig 32). Lesions of the occipital cortex may result in mind blindness (optic agnosia) the patient sees objects but cannot recognise them. Visual hallucinations may also occur. Wernicke's hemiopic pupillary reaction is described as an aid to determine whether a lesion of the visual path is situated before or behind the primary visual centre of the superior corpus quadrigeminum. A narrow beam of light is cast into the eye by an ophthalmoscope mirror on to the blind half of the

retina. If the pupil contracts the light reflex fibres are not involved and so the lesion is situated behind the primary visual centre, *i.e.*, in the internal capsule, optic radiation or cortex. In practice it is very difficult to carry out this test.

*Amaurosis* is a term used to indicate blindness. Uræmic amaurosis may occur in the course of acute or chronic nephritis. The patient suddenly becomes blind in one or both eyes. The pupils are dilated but react to light. The discs and fundi are normal apart from any changes associated with the nephritis. There is usually spontaneous recovery in the course of a day or so (see p. 400).

*Amblyopia* indicates some dimness of vision. In hysterical amblyopia the pupils and fundi are normal, but the fields of vision show a spiral restriction during testing, either from within outwards, or from without inwards, according to the manner in which the test is conducted. Hemianopia may also occur in hysteria or in migraine.

### The III, IV, and VI Nerves

#### (The Oculo-motor Nerves)

*Anatomy.* The III nucleus lies in the mid-brain at the level of the superior corpora quadrigemina in the floor of the Sylvian aqueduct. Some fibres cross, joining the III nerve of the opposite side. The nerve

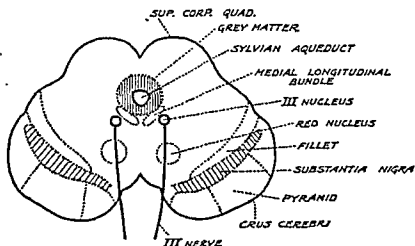


FIG. 33. DIAGRAM OF THE MID-BRAIN AT THE LEVEL OF THE SUPERIOR CORPORA QUADRIGEMINA.

leaves the brain on the ventral surface near the mid-line on the inner side of the crus, and passes through the outer wall of the cavernous sinus to enter the orbit through the sphenoidal fissure (see Fig. 33). It divides into a superior branch supplying the levator palpebræ superioris and the superior rectus muscles, and an inferior branch distributed to the inferior and internal recti and the inferior oblique muscles. It also gives fibres to the ciliary ganglion from which the short ciliary nerves run to the ciliary muscle and the constrictor of the pupil. The IV

nucleus is situated in the mid brain at the level of the inferior corpora quadrigemina, in the floor of the Sylvian aqueduct. The IV nerve decussates in the valve of Vieussens and leaves the brain on the opposite side on the dorsal surface (see Fig 34). It passes forwards around the crus cerebri and runs in the outer wall of the cavernous sinus below the III nerve. Passing through the sphenoidal fissure it ends in the superior oblique muscle. The VI nucleus is situated close to the VII nucleus in the lower part of the pons, in the floor of the fourth ventricle. It leaves the brain on its ventral surface at the lower border of the pons and runs in the inner wall of the cavernous sinus just external to the internal carotid artery. It passes through the sphenoidal fissure to the orbit and supplies the external rectus muscle. The ophthalmic branch of the V nerve also runs in the outer wall of the cavernous sinus, the nerves

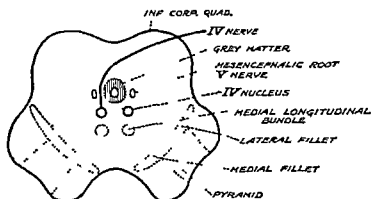


FIG 34. DIAGRAM OF THE MID BRAIN AT THE LEVEL OF THE INFERIOR CORPORA QUADRIGEMINA.

being in the following order from above downwards III, IV, ophthalmic branch of V, and VI (see Fig 28).

**The Oculo-motor Muscles** The external muscles of the eye produce movements of the eyeball in three directions: 1 Vertical, an upwards or downwards movement; 2 Horizontal, an inwards or outwards movement; 3 Torsion or wheel motion, the vertical meridian being rotated inwards or outwards.

The movements effected by the muscles are as follows: *The external rectus muscle* This moves the eye outwards. *The internal rectus muscle* This moves the eye inwards. *The superior rectus muscle* This moves the eye upwards and inwards and rotates or twists it inwards. *The inferior rectus muscle* This moves the eye downwards and inwards and twists it outwards. *The superior oblique muscle* This moves the eye downwards and outwards and twists it inwards. *The inferior oblique muscle* This moves the eye upwards and outwards, and twists it outwards. *Diplopia* or double vision results from paralysis of the external ocular muscles. It results from the axes of the eyeballs not being parallel when an object is looked at. The patient therefore sees two objects, as the images do not fall on corresponding parts of the retinae. The true image is the one seen by the sound eye. It is

diplopia is present on looking downwards, it is crossed, the false image being below and tilted towards the true image *Paralysis of the external rectus* There is inability to move the eye outwards. The diplopia is homonymous, the false image is on the same level as the true one, and on the same side of it as is the lesion. Thus with a left external rectus paralysis the patient complains of diplopia when he looks to the left. There is an internal strabismus, the affected eye being turned inwards by the unopposed action of the internal rectus. The head may be kept a little turned to the affected side. Secondary deviation may be detected in the sound eye. Thus if the patient is asked to look outwards with the affected eye, and the sound eye is screened, the sound eye moves inwards to a greater degree than the affected eye moves outwards. This is because a greater stimulus than normal is given to the affected eye to try and cause it to move, and a correspondingly greater contraction takes place in the internal rectus of the sound eye, which normally works in unison with the external rectus of the other eye. The farther the test object is moved to the affected side, in determining the diplopia, the greater is the distance between the true and false images. The patient also misjudges distances on looking to the affected side, the object appearing farther away than it really is. *Paralysis of the internal rectus* There is inability to move the eye to the affected side. There is external strabismus. The patient has crossed diplopia on looking inwards, the false image being on the opposite side, but on the same level as the true one. *Paralysis of the inferior oblique* There is weakness in moving the eye upwards and outwards, and in rotating outwards. There is diplopia on looking upwards. The patient may elevate the head and then look downwards to avert the diplopia which is homonymous, the false image being situated above and tilted away from the true image. *Paralysis of the superior oblique* There is weakness in moving the eye downwards and outwards, and in rotating it inwards. The patient complains of diplopia and giddiness on looking down, as on descending a flight of stairs. The diplopia is homonymous, the false image is below and tilted towards the true image.

**Lesions of the Oculo-motor Nerves** Lesions may occur in three portions of the motor tract from the cerebral cortex to the muscles of the eye. 1 Supranuclear lesions in the cortex, corona radiata, internal capsule or mid brain. 2 Nuclear, affecting the III, IV or VI nuclei. 3 Infranuclear, affecting the nerves themselves.

### Supranuclear Lesions

**Etiology.** These may result from vascular disturbances, such as hæmorrhage, thrombosis or embolus, or from the pressure of a tumour. The cortical centre for eye movements is thought to be situated in the region of the second frontal gyrus.

**Clinical Findings** Both eyes are affected. There is usually a disturbance of conjugated lateral movements. In bilateral internal capsular lesions, or in unilateral mid brain lesions in the region of the superior corpora quadrigemina or Sylvian aqueduct there may be disturbance of conjugated vertical movements of both eyes. There is

usually an associated hemiplegia. Thus with a right-sided lesion there is paralysis of the left side of the body, with conjugate deviation of the eyes. The direction of this deviation depends upon whether the lesion is an irritative or a paralytic one. With an irritative lesion the eyes look away from the lesion, *i.e.*, if the lesion is on the right side of the brain, the eyes are deviated to the left. The reverse holds good with a paralytic lesion. This is explained by reference to the diagram (see Fig. 37). The cortical lesion affects the VI nucleus on the opposite side, and the VI nucleus is connected to, and works synergically with the portion of the III nucleus of the opposite side which supplies the internal rectus muscle.

**Treatment.** This varies with the cause, as for cerebral vascular lesions or tumours (see pp. 305, 306, 314).

### Nuclear Lesions

**Etiology.** The oculo-motor nuclei may be stimulated or paralysed by such lesions as a hæmorrhage, thrombosis or embolus, a mid-brain

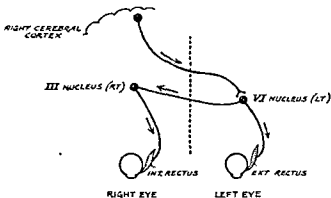


FIG. 37. DIAGRAM OF THE MECHANISM OF CONJUGATE DEVIATION OF THE EYES.

or pontine tumour. A cerebellar tumour may affect the VI nerve nucleus by indirect pressure. Other lesions include those produced by acute encephalitis occurring as a complication of measles, influenza, scarlet fever or small-pox, encephalitis lethargica, tabes dorsalis, syringomyelia, chronic poliomyelitis, disseminated sclerosis, and botulism. A nuclear lesion may also occur in myasthenia gravis. In Veronal poisoning the external ocular muscles may be affected by a nuclear lesion.

**Clinical Findings.** Nuclear ophthalmoplegia may appear as an acute or chronic disease. In the acute variety there is often headache and vomiting, with a raised temperature. Both eyes are affected, usually groups of muscles, but not all the muscles are involved. There may also be internal ophthalmoplegia, the pupil being moderately dilated, and showing no response to light or to accommodation; usually, however, the intraocular muscles are not affected. An irritative nuclear lesion causes a conjugate deviation of the eyes, so that they look towards the side of the lesion, and a paralytic lesion has the converse

effect. This is the reverse of what occurs in a supranuclear lesion, and is explained by the diagram (see Fig. 37). If the pyramidal tract is affected, there is hemiplegia on the side of the body opposite to the lesion (Weber's syndrome). If the rubro spinal tract is also involved there are involuntary tremors of the opposite side of the body. Chronic nuclear paralysis is more often met with. The onset of the symptoms is insidious. There is usually conjugate deviation of the eyes, the patient not being able to turn the eyes in one lateral direction. It is often difficult to differentiate between single nuclear lesions and infranuclear lesions when only one muscle is affected. If in addition to some of the muscles supplied by the III nerve the orbicularis palpebrarum muscle is also paralysed, the lesion is probably a nuclear one. Lesions of the VI nucleus are usually accompanied by a VII nerve nuclear lesion as well. A VI left nuclear lesion causes conjugate paralysis to the left side, but if the left eye is covered, the right eye can be moved inwards, and both eyes will converge.

*Jaw-winking* may occur in cases of congenital ptosis. When the jaw is depressed to the opposite side by contraction of the external pterygoid muscle of the same side, the impulse, passing along the V nerve, causes the upper lid on the same side to be twitched up, the paralysed levator palpebræ superioris contracting. It is usually unilateral.

### Infranuclear Lesions

**Etiology.** The oculo motor nerves may be affected at different parts of their course. 1. *In the brain.* The lesions here are usually vascular or due to tumours. Increased intracranial pressure, associated either with supra- or infratentorial growths, by stretching the nerve, may cause VI nerve paralysis. This constitutes a false localising sign of a VI nerve lesion. 2. *Between the pons and the orbit.* The lesion may be meningitis, often syphilitic, a gumma, an intracranial tumour, neuritis due to diphtheria, diabetes mellitus, or less often to lead, alcohol or nicotine toxic neuritis from septic foci elsewhere, fractured base of the skull, cavernous sinus thrombosis, aneurysm or atheroma of the internal carotid or posterior communicating artery, and subarachnoid hæmorrhage. The VI and V nerves may be affected in acute mastoiditis in children, probably due to a localised meningitis. *In Gradenigo's syndrome* there is weakness of the external rectus muscle on one side, due to involvement of the VI nerve by inflammation of the apex of the petrous bone in association with otitis media. The patient is ill with fever, and complains of a headache on the same side. Ophthalmoplegic migraine may be due to unilateral distension of the lateral ventricle. In the sphenoidal fissure, acute periositis may cause infranuclear paralysis, a condition comparable with peripheral facial palsy. There is then pain in the eye with partial or total ophthalmoplegia and proptosis. The ophthalmic and maxillary divisions of the V nerve may also be involved with cutaneous anaesthesia over their area of distribution. Overgrowth of bone, as in leontiasis ossea, may cause pressure paralysis. 3. *In the orbit.* The nerves may be involved in

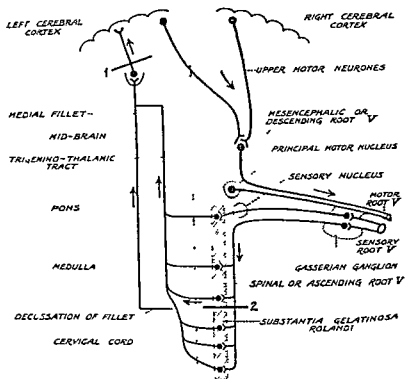


FIG. 38. DIAGRAM OF THE CENTRAL PATH OF THE V NERVE.

Lesion at 1 causes right hemianæsthesia.

Lesion at 2 causes alternate hemianæsthesia, right side of face, left side body, arm and leg

The V nerve, after leaving the pons, passes forwards over the petrous bone, where the Gasserian ganglion is situated on the sensory part of the nerve, the motor part lying beneath this ganglion. Three main nerves emerge from the Gasserian ganglion, named the ophthalmic or first, the maxillary or second, and the mandibular or third division. The motor nerve is attached to the third division. The *ophthalmic branch* runs in the outer wall of the cavernous sinus and passing through the sphenoidal fissure to the orbit, terminates in the lachrymal, frontal and nasal nerves. In the cavernous sinus it gives a branch to the dura mater. The lachrymal nerve supplies the lachrymal gland, the conjunctiva, cornea, and the outer canthus of the eye. The frontal nerve divides into the supra orbital and supra trochlear nerves, and supplies the skin on the front half of the scalp and forehead, the root of the nose and inner canthus of the eye, the upper eyelid and the frontal sinus. The nasal nerve supplies the mucous membrane of the upper part of the nose, and the skin of the lower part and tip of the nose.

The *maxillary branch* runs along the lower part of the cavernous sinus, enters the foramen rotundum and sphenomaxillary fossa, and traverses the orbit to appear on the face through the infra orbital foramen. In the skull it gives a recurrent branch to the dura mater. In the infra orbital canal it supplies branches to the teeth of the upper jaw. Its cutaneous branches are distributed to the face between the lower eyelid, side of the nose and upper lip. Branches are also given to the mucous membrane of the hard palate and the antrum of Highmore. The *mandibular branch* leaves the skull through the foramen ovale, and gives a recurrent branch to the dura mater. The motor fibres supply the following muscles: The masseter, the temporal, the external and internal pterygoids, the mylohyoid, the anterior belly of the digastric, the tensor tympani and the tensor palati. The auriculotemporal branch supplies cutaneous branches to the temple and scalp, and to the external auditory meatus and upper part of the pinna. This nerve also gives branches to the parotid gland and the temporo-mandibular joint. The lingual branch supplies the tactile filiform papillae in the anterior two-thirds of the tongue. The chorda tympani nerve joins the lingual nerve and conveys taste sensations from the tongue to the VII nerve, and then to the glosso-pharyngeal nucleus (see p. 380). The inferior dental branch supplies the teeth of the lower jaw, and emerging through the mental foramen innervates the skin over the chin and lower lip. The mucous membrane of the mouth, gums and Eustachian tube is also supplied by this division of the V nerve.

**Lesions of V Nerve** The following varieties of lesions may occur.

- Supranuclear lesions** The cortical centres for the muscles innervated by the V nerve are situated in the lower third of the precentral convolution. These centres are bilateral, each centre sending fibres to both V nuclei, and so both centres must be put out of action to cause an upper motor neurone paralysis of the masticatory muscles. Lesions may occur here, in the corona radiata, internal capsule, or mid brain. They are rare and include meningeal and vascular disturbances and tumours. In epilepsy, hydrophobia and post-encephalitic syndromes



supranuclear disturbances of the V nerve may occur. In paralysis agitans there is probably a lesion in the corpora striata disturbing the fibres in the internal capsule. In strychnine poisoning and tetanus, the lesion is probably situated in the synapses connecting the upper motor neurones with the nuclear cells.

2. *Nuclear Lesions.* These are situated in the pons and include hæmorrhage, tumour, gumma, polioencephalitis, disseminated sclerosis, bulbar paralysis, amyotrophic lateral sclerosis and syringomyelia.

3. *Infranuclear Lesions.* The V nerve may be affected at the base of the brain by meningitis or fracture of the base of the skull, or its branches may be involved by a tumour, thrombosis of the cavernous sinus, an aneurysm of the internal carotid artery and cellulitis of the orbit. The Gasserian ganglion may be inflamed, or the branches of the V nerve involved in a neuritis, such as occurs in diphtheria.

**Clinical Findings.** Disturbances of the V nerve may affect chiefly the motor or sensory fibres or combined lesions may occur. 1. *Motor Lesions.* These may be irritative or paralytic. (a) *Irritative lesions.* Tonic or clonic contractions of the masticatory muscles occur. The former are known as trismus, and may be present in tetanus, epilepsy, tetany, or strychnine poisoning. Clonic spasms are seen with rigors and paralysis agitans, etc. Nocturnal jaw-grinding is a functional nervous disturbance, and thought to be an indication of mental protest. (b) *Paralytic lesions.* Supranuclear lesions when bilateral are characterised by paralysis of the lower jaw. The patient can neither close the mouth, protrude the jaw, nor move it from side to side. Food tends to drop from the mouth. There is no wasting of the muscles and no reaction of degeneration. Nuclear lesions are often bilateral, with a flaccid paralysis of the masticatory muscles on both sides. Infranuclear lesions are usually unilateral. There is often wasting and weakness of the muscles of mastication on one side of the face. When the patient opens his mouth the lower jaw is pushed to the paralysed side by the action of the opposite external pterygoid muscle. This is best judged by the position of the central incisors of the upper jaw when the mouth is open and closed. There is inability to move the jaw laterally to the sound side, and on opening the mouth the condylar process of the lower jaw can be felt not to move forwards on the affected side. When the patient is asked to bite, it can be felt that the masseter and temporal muscles do not contract, and some flaccidity of the floor of the mouth may be found on pressing below the chin. The jaw-jerk is absent, and the affected muscles show a reaction of degeneration.

2. *Sensory Lesions.* (a) *Loss of Sensation.* An upper motor neurone lesion affecting the posterior part of the internal capsule may cause loss of sensation of the face, the arm, body and leg on the opposite side of the body (crossed hemianæsthesia). A lesion of the spinal root of the V nerve and the mesial fillet on the same side will cause "alternate hemianæsthesia," the face on one side, and the arm, body and leg on the other side being affected (see Fig. 38). Lower motor neurone lesions may affect individual branches of the V nerve. Thus in periostitis of the orbit the ophthalmic division may be affected with certain oculo-

motor nerves as described on p 366 There is then blunting of sensation in the cutaneous area of this nerve around the orbit and forehead (see p 368), and the corneal and conjunctival reflexes may be abolished with diminished tear secretion When the maxillary and mandibular branches are involved, in addition to the cutaneous anæsthesia, the patient cannot feel his tongue and cheek normally, and is apt to bite it, and in drinking from a glass he can only feel half the glass touching his lip There may also be diminished secretion of saliva on one side, the tongue is dry and furred on the affected side, and using a tuning fork of low pitch, there may be loss of appreciation for low notes Anæsthesia is found in the cheek and gums Smell may be diminished on one side, owing to dryness of the nasal mucous membrane Taste is not affected unless the chorda tympani nerve is also involved Segmental blunting or loss of sensation may also occur, limited to trigeminal dermatomes around the mouth The teeth on the affected side may fall out Facial or corneal herpes and corneal ulceration may also be a manifestation of lesions of the sensory division of the V nerve or of the Gasserian ganglion

(b) *Trigeminal Neuralgia* This may be of a minor degree with pain situated in the region of certain branches of V nerve. Thus there may be supra orbital neuralgia associated with frontal sinusitis Tenderness may be found at the supra orbital notch Headache may be due to involvement of the branches supplying the dura mater The severe type of trigeminal neuralgia is known as *tic douloureux* This is an affection of middle age or later, but it is occasionally met with in children There are periodical paroxysms of agonising pain in the face, especially in the areas supplied by the second and third divisions of the V nerve The pain may spread to the neck A paroxysm lasts for a few seconds or minutes, and tends to recur frequently During the attack the face may become flushed and moist with perspiration, and there may be watering of the eyes and mouth and twitching of the facial muscles The attack may be provoked by the slightest stimulus, such as a draught, washing or shaving, cleaning the teeth or eating

*On Examination* Tender spots may be found over the infra orbital or mental foramina

*Treatment* In all cases of trigeminal neuralgia the cranial sinuses should be investigated, frontal sinusitis may result in supra orbital or supra trochlear neuralgia The pain is best relieved by local heat, which can be conveniently applied by a rubber hot water bottle A nasal spray should also be used containing some preparation, such as the Chlorotone Inhalant Dental extraction rarely relieves the pain, and should not be advised unless in obviously diseased tooth is present In *tic douloureux* the patient should have complete rest in bed for several weeks, and for the pain the following mixture may be given *Infusio* *gelsenu* m 10 *sod. salicyl* gr 5 *sod bicarb* gr 20, *sod brom* gr 10, *syr aurant* m 20 *aquam ad* fl oz 1 fl oz 1 tds pc In severe and protracted cases relief may in some cases be obtained for several months by injecting the Gasserian ganglion or its branches with m 10 to 15 of 80% alcohol through the foramen ovale or foramen rotundum

This is an operation which should only be performed by a specialist. It may be repeated later if the pain recurs. In very severe cases ganglionectomy, or resection of the posterior nerve root inside the skull, may be necessary to relieve the pain. Even then the patient may still feel pain, although the sensory fibres are cut off from the brain. This is comparable with the sensation which may be felt in the toes after a leg has been amputated.

3. *Trophic Lesions.* It has been mentioned above that corneal ulceration may follow lesions of the first division of the V nerve. If the eye is protected by a shade from the irritation of dust, etc., these lesions do not occur. They are probably mechanically produced, the irritants not being removed by blinking, as they are not felt. Facial hemiatrophy is a peculiar condition in which there is wasting in structures supplied by the V nerve on one side. The cause is not known, but it may be due to any nuclear or infranuclear trophic disturbance of the V nerve, and is at times associated with osteitis of the jaw, or it may follow erysipelas. The bones, cartilages and soft tissues are affected, chiefly in the areas supplied by the second and third divisions of the V nerve. One side of the face is smaller than the other. The skin becomes thin and the subcutaneous fat disappears; there may also be atrophy of half of the tongue. There are no sensory changes, the muscles are not paralysed, and there is no reaction of degeneration.

### The VII Nerve (The Facial Nerve)

**Anatomy.** The upper neurone has its cells of origin in the cerebral cortex, in front of the Rolandic fissure, in the lower third of the precentral gyrus. The fibres pass through the internal capsule, near the genu, and run down the mid-brain in the pyramidal tract to the lower part of the pons. When they reach the level of the nucleus of origin of the VII nerve they cross the mid-line and terminate around the cells of origin of the VII nerve. A few fibres probably end around the homolateral nucleus. The VII nerve is partly motor and partly sensory. The motor nucleus is situated in the lower part of the pons, close to the VI nucleus. The VII nerve, after coursing around the VI nucleus, passes out from the antero-lateral aspect of the lower part of the pons in the cerebello-pontine angle, and runs close to the VIII nerve, to enter the internal auditory meatus and the Fallopian aqueduct of the temporal bone. The geniculate ganglion is situated on the VII nerve at this part of its course, and, shortly after, the nerve to the stapedius is given off. The chorda tympani nerve, conveying taste sensations from the anterior two-thirds of the tongue, joins the VII nerve while it is still in the bone. The VII nerve emerges from the skull through the stylo-mastoid foramen. In the neck it gives off the posterior auricular nerve to the intrinsic muscles of the pinna and to the occipital part of the occipito-frontalis muscle, and nerves to the stylo-hyoid muscle and to the posterior belly of the digastric muscle. The VII nerve then divides into the temporo-facial and cervico-facial branches.

The former supplies the *corrugator supercilii*, the *frontalis*, the *orbicularis palpebrarum* and the zygomatic muscles, and the latter the buccinator, *orbicularis oris* and *platysma* muscles (see Fig 39). The sensory part of the nerve is considered to be formed by the *pars intermedia* of Wrisberg, which runs from the geniculate ganglion to the *tractus solitarius* portion of the glossopharyngeal nucleus in the medulla. The taste fibres in the chorda tympani nerve pass from the geniculate ganglion to the nerve of Wrisberg. The geniculate ganglion is also joined by the large, the small and the external superficial petrosal nerves which carry sympathetic and taste fibres. Further sensory fibres conveying deep pain and pressure sensations run in the peripheral divisions of the facial nerve, and pass through the geniculate ganglion to the *pars intermedia* of Wrisberg.

**Lesions of the VII Nerve** 1 *Supranuclear* The lesion may be situated in the cerebral cortex (lower part of the precentral gyrus), the corona radiata, genu of the internal capsule, or in the suprapontine region. The lesions include a cerebral tumour, abscess or vascular lesion such as a hæmorrhage, thrombosis or embolus. 2 *Nuclear* In the pons. A tumour such as a glioma, a vascular lesion, encephalitis lethargica, disseminated sclerosis, bulbar paralysis, poliomyelitis, and *tabes dorsalis*. 3 *Infranuclear* (a) *At the base of the brain* Tumours of the cerebello pontine angle, basal meningitis, a gumma, aneurysm of the basilar artery, or a fractured base. (b) *In the temporal bone* Caries associated with otitis media, toxic neuritis, an operation on the mastoid. (c) *In the stylo mastoid foramen* Fibrositis of the process of the posterior part of the parotid sheath which enters the foramen, associated with exposure to cold. (d) *In the face* A tumour or inflammation of the parotid gland, as in uveo-parotid tuberculosis (see p 15). Injury from a wound or from forceps at birth. Neuritis due to such causes as alcohol, diphtheria, diabetes mellitus or leprosy.

**Clinical Findings** 1 *Supranuclear Lesions* (a) *Irritative* With cortical lesions irritating the motor centre unilateral facial contractions may occur on the opposite side of the face. There are often associated movements of the eyes and tongue. In *dementia paralytica* peri oral fibrillary tremors may be noted. (b) *Paralytic* There is paralysis chiefly affecting the lower part of the face of the opposite side, the upper part is much less likely to be paralysed, owing to the bilateral representation of the cortical centres in respect to their supply to the upper part of the face. Thus although the mouth appears drawn over to the sound side and the *orbicularis oris* muscle is paralysed on the affected side, the patient can close both eyes and wrinkle his forehead. The paralysed muscles do not atrophy and there is no reaction of degeneration. Taste is not affected. In addition there is frequently hemiplegia, the arm, body and leg being paralysed on the same side as the face, when the lesion is a capsular one. Although the patient cannot contract the facial muscles voluntarily on one side, he may do so under the influence of emotion. Thus a joke may cause him to smile with both sides of his face, in fact the facial contraction may be exaggerated on the paralysed side. This is not the case in infranuclear lesions.

2. *Nuclear Lesions.* There is paralysis of the upper and lower half of the face on the same side as the lesion. The muscles atrophy and show the reaction of degeneration. Further, there is often paralysis of the external rectus muscle of the eye on the same side, due to involvement of the contiguous VI nucleus. If the pyramidal tract is also affected there is spastic hemiplegia of the opposite side of the body.

3. *Infranuclear Lesions.* (a) *Irritative.* Severe facial spasms may occur, functional tremors of the eyelids and facial tics are also of this nature. (b) *Paralytic* (Bell's palsies). The clinical results vary according to the site at which the lesion is situated, as described below: (a) *Between the pons and geniculate ganglion.* There is paralysis on the same side of the upper and lower half of the face, of the lower motor neurone type (see below, section (d)). The VIII nerve may also be involved with deafness or tinnitus, or the V nerve with paralysis or sensory disturbances, and if the nerve to the stapedius is affected there may be hyperacusis to sounds of low or high pitch. Taste is not affected unless the nerve of Wrisberg (pars intermedia) is also involved. (b) *At the geniculate ganglion.* There is paralysis on the same side of the upper and lower half of the face, and herpes located to the ear and external auditory meatus (herpes oticus or Ramsay Hunt syndrome) with pain, may result from inflammatory lesions. The chorda tympani nerve fibres may also be affected with loss of taste on the anterior two-thirds of the tongue on the same side. (c) *Between the geniculate ganglion and the chorda tympani nerve.* There is paralysis on the same side of the upper and lower half of the face (see below, section (d)). There is loss of taste on the anterior two-thirds of the tongue on the same side. If the lesion is proximal to the junction of the nerve to the stapedius there may be tinnitus and hyperacusis for notes of high or low pitch. Salivary secretion may be diminished on the same side. (d) *Peripheral to the junction of the chorda tympani nerve.* (The usual type of Bell's palsy.) This may follow exposure to cold or sitting in a draught near an open window. Both sexes are equally affected. The patient is usually an adult over the age of 20. He may notice some pain or tenderness behind the ear for a day or so, and then suddenly finds that he cannot move one side of the face normally and this side of the face may feel stiff. He may also find that when he eats he tends to bite his cheek or lower lip on one side, and the food may collect between the cheek and teeth on this side. Tears may also run down the cheek on the affected side, due to paralysis of the orbicularis palpebrarum muscle.

*On Examination:* The paralysed side of the face appears flat and expressionless, the naso-labial fold is less apparent. The lower lid may be everted in one eye, and the corner of the mouth droops. Some swelling or tenderness may be found in the parotid region. There is paralysis of the muscles of the upper and lower parts of the face. These are tested as follows: The patient is asked to shut his eyes; he cannot close the eye on one side (lagophthalmos), and on trying to do so the eyeball moves upwards and either slightly inwards or outwards, owing to a

connection between the VII and a portion of the III nucleus. If the patient is observed during sleep the affected eye may be nearly closed. He is asked to frown and to raise his eyebrows and is unable to do so on the paralysed side. The lower part of the face is tested by asking the patient to show his teeth, the angle of the mouth is drawn outwards on the sound side and does not move out on the paralysed side. He is then asked to blow out his cheeks, to smile and to whistle. He cannot do so on the paralysed side. Spontaneous emotions also fail to provoke a contraction of the oral muscles on the paralysed side, in contrast to that which may occur in organic supranuclear lesions with hemiplegia and facial paralysis (see p 372). Paralysis of the platysma muscle may sometimes be detected by making the patient depress his chin against the resistance of the examiner's hand, when it may be seen, by looking at the skin under the chin, that the platysma does not contract on one side. The affected muscles atrophy and may give a reaction of degeneration. In the course of a few days the lesion may spread up to involve the chorda tympani nerve. Taste is then lost on the affected side of the anterior two-thirds of the tongue. It is tested for as follows. The patient is given a card on which the words 'Sweet Salt Sour Bitter' are written and he is told to point to the word representing the sensation he experiences when the test substance is applied to the tongue. He must not withdraw his tongue into his mouth until he has indicated the sensation. Sugar, salt, citric acid and quinine are employed as the test substances. A small portion is applied in turn to the outer side of the anterior part of the tongue and gently rubbed in with cotton wool. If the inflammation spreads still further up the VII nerve above the level of the junction of the nerve to the stapedius, there may be tinnitus and hyperacusis to notes of high or low pitch.

**Differential Diagnosis.** There is usually no difficulty in diagnosing a Bell's palsy: the distinctions between upper and lower motor neurone lesions described above should be remembered. In facio scapulo humeral myopathy usually both sides of the face are affected and also other muscles of the body which differentiates it from a double facial paralysis. In facial hemiatrophy there is wasting but not paralysis of the muscles of one side of the face.

**Course and Complications.** The paralysis may disappear in a week or so, or persist for nearly 2 years. Recovery usually is first apparent in the upper half of the face. Subsequent twitching or contractures of the affected muscles may occur, when the recovery is not complete. Thus the eye on the affected side may be narrow, or the corner of the mouth drawn outwards. 'Crocodile tears' may also be expected in cases of incomplete recovery. When the patient masticates and salivates tears flow from the eye on the affected side as the result of stimulation of the lachrymal gland.

**Prognosis.** The prognosis in Bell's palsy depends upon the cause. In the usual type due to fibrositis or periostitis there is recovery in about 80% of cases. The electrical reactions are of help in giving a prognosis. If the faradic and galvanic responses of the paralysed muscles are normal after 2 weeks, recovery will be rapid, a matter

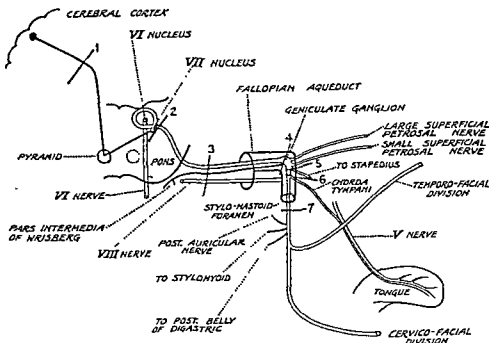


FIG. 39. DIAGRAM OF THE VII NERVE.

*Lesion at 1.*—Upper Motor Neurone. Paralysis of orbicularis oris. Often hemiplegia on the same side. No muscular atrophy and no R. D.

*Lesion at 2.*—Nuclear. Orbicularis palpebrarum and orbicularis oris paralysed. Muscular atrophy and R. D. Hemiplegia may occur on the opposite side. VI nerve often paralysed.

*Lesion at 3.*—Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris. VIII nerve also affected. Taste affected if nerve of Wrisberg is involved.

*Lesion at 4.*—Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris. Auricular herpes. Taste may be lost on anterior two-thirds of tongue.

*Lesion at 5.*—Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris. Hyperacusis and loss of taste in anterior two-thirds of tongue.

*Lesion at 6.*—Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris. Loss of taste in anterior two-thirds of tongue.

*Lesion at 7.*—Lower motor neurone lesion of orbicularis palpebrarum and orbicularis oris. No loss of taste.

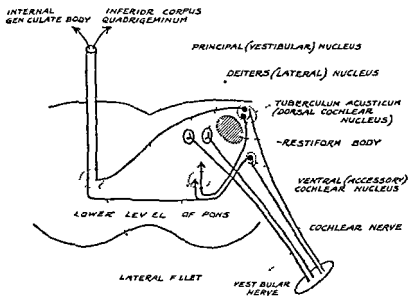


FIG 40 DIAGRAM OF THE VIII NERVE NUCLEI



of a few weeks. If there is no response to faradism and the galvanic response shows a reaction of degeneration, recovery will not occur for several months. If no response is obtained to faradism or to galvanism, there is no likelihood of recovery. If the paralysis is due to other causes, such as a tumour, gumma or meningitis, the prognosis must vary with the nature of the lesion and the possibility of removing it by appropriate treatment.

**Treatment.** In the inflammatory type of case the patient should be kept in a warm room during the initial stages until all pain and tenderness have gone. Heat should be applied locally over the stylo-mastoid foramen, as by Antiphlogistine (cataplasma kaolini B.P.) or by fomentations. The bowels should be opened daily, and a mixture given containing: Pot. iod. gr. 3, sod. salicyl. gr. 10, sod. bicarb. gr. 20, syr. aurant. m. 20, aq. ad fl. oz. 1. Fl. oz. 1 t.d.s. p.c. A wire splint, protected by rubber over the pressure points, should be worn which curves round the ear and into the corner of the mouth on the affected side. This prevents the mouth being drawn over to the sound side. After a week, massage should be given daily to the affected muscles. In all cases the Wassermann reaction should be determined, and, if positive, a course of anti-syphilitic treatment should be given (see p. 248). Some surgeons advise early operation, either by decompression of the descending portion of the nerve, or by placing in the Fallopian canal a prepared nerve graft, obtained from a cutaneous nerve of the thigh (Ballance-Duel nerve graft). In cases in which there is no recovery after 2 years, the operation of nerve anastomosis should be considered. The peripheral end of the severed facial nerve is sutured to the proximal end of the severed hypoglossal or spinal accessory nerve. This should only be decided after consultation with an expert.

**Bilateral Facial Paralysis.** This may be congenital, and there is often paralysis of the muscles supplied by the oculo-motor nerves as well. Other causes include double otitis media, basal gummatous meningitis, an aneurysm of the basilar artery, diphtheria, leprosy and alcoholic neuritis. Both sides of the face are then expressionless.

### The VIII Nerve

#### (The Auditory Nerve)

**Anatomy.** The VIII nerve consists of a cochlear and a vestibular division. The former is concerned with hearing, and the latter with equilibrium. The cochlear nerve fibres arise from cells in the spiral ganglion, situated in the central pillar of the cochlea, their peripheral terminations ending in connection with the hair cells of the organ of Corti. Centrally the cochlear nerve passes to the brain through the internal auditory meatus, running below the VII nerve. It enters the lower border of the pons on the lateral side of the restiform body, and terminates around cells of the dorsal nucleus or tuberculum acusticum and the ventral or accessory cochlear nucleus. A fresh relay of fibres conducts the impulses across the mid-line in two strands, a dorsal or

stræ acusticæ and a ventral or corpus trapezoides. These unite to form the lateral fillet. Some of the fibres from the cochlear nuclei also run to the lateral fillet of the same side. The lateral fillet ascends the mid brain and terminates in the lower auditory centres, the internal geniculate body and the inferior corpus quadrigeminum. The internal geniculate bodies are connected by Gudden's commissure. A further relay conveys the impulses through the posterior part of the internal capsule to the higher auditory centres in the superior temporal gyrus of the cerebral cortex.

The cells of origin of the vestibular nerve are in the ganglion of Scarpa which lies in the internal auditory meatus. Their peripheral terminations are in the semicircular canals and the otolith organs (the utricle and saccule). The fibres from the semicircular canals convey sensations of movement (kinetic impulses), whereas those from the otolith organs transmit sensations of position (static impulses). Centrally the fibres pass with the cochlear division and enter the lower part of the pons between the restiform and olivary bodies, terminating around the cells of the principal or vestibular, and Deiters' or the lateral nucleus. The upper part of Deiters' nucleus constitutes Bechterew's nucleus (see Fig. 40). The further course of the vestibular path is rather open to discussion. Fibres pass from Deiters' nucleus up the pons and mid brain in the medial longitudinal bundle. In this way connection is made with the nuclei of the oculo motor nerves, thus establishing relationship between equilibrium and eye movements. Impulses are also carried downwards in the medial longitudinal bundle to connect with the VI nucleus and establish a relationship with equilibrium and head movements. Fibres also descend the cord in the vestibulo spinal tract whereby a connection is made between the sense of equilibrium and the tone of skeletal muscles. Some fibres of the vestibular nerve run direct to the cerebellum by the inferior cerebellar peduncle, and fibres also pass from the cerebellum by Edinger's tract in the inferior cerebellar peduncle to Deiters' nucleus (see Fig. 31).

**Lesions of the VIII Nerve.** The VIII nerve may be affected by tumours of the cerebello pontine angle pressing on or growing from the nerve itself, or by basal meningitis due to syphilis or cerebro spinal fever. Neuritis may be due to toxins of typhoid or scarlet fever, to syphilis, or to such substances as alcohol, tobacco, quinine or salicylates. Atrophy may occur in *tabes dorsalis* or in *disseminated sclerosis*. The nerve may be injured in a fracture of the base of the skull or by disease of the petrous bone. Inflammation may affect it in otitis media, labyrinthitis or Eustachian catarrh. It may be involved in a vascular lesion, such as a hæmorrhage or thrombosis, and by a leukæmic infiltration. Central lesions are rare. A bilateral lesion of the superior temporal gyrus may cause deafness, or word deafness may result from an unilateral lesion affecting the left superior temporal convolution. In hysteria there may be complete deafness.

**Clinical Findings.** Lesions of the VIII nerve may result in deafness, tinnitus or vertigo. **Deafness.** When examining a patient who complains of deafness, the following tests may be required. 1. The external

ear is examined for wax or other foreign bodies. 2. The distance at which the ticking of a watch can be heard is determined for each ear. 3. The tuning-fork test. A tuning-fork, with vibration of 256 per second, middle C on the piano (C<sup>4</sup>) is used. It is made to vibrate and then placed on the centre of the top of the skull, on the centre of the forehead between the eyes, or on the point of the chin. The patient is asked in which ear he hears the vibrations better. Normally they should be heard equally in both ears, and when one ear is closed, they are heard louder on that side (Weber's test). The vibrating fork is then placed over one mastoid process, and when the vibrations can no longer be heard it is held close to the external ear. Normally the vibration can now be heard again for a short period (Rinne's test). The following tests help to differentiate between nerve deafness in which there is a lesion of the internal ear (cochlea), or of the auditory nerve, and obstructive deafness in which the trouble lies in the middle or outer ear. In *nerve deafness* it may be found that the deafness is chiefly for notes of high pitch, as shown by the use of tuning-forks of different vibration rates. When the tuning-fork is placed on the vertex, it is not heard on the affected side (negative Weber test). Usually aerial conduction is better than bone conduction (positive Rinne test). In *obstructive deafness* the patient may notice that he hears better in a noise, as in traffic (*paracusis willisii*). The vibrating tuning-fork is heard louder on the deaf side (positive Weber test), and a high-pitched tuning-fork is heard better than a low-pitched one. Aerial conduction is not better than bone conduction (negative Rinne test).

*Tinnitus aurium.* The chief symptom complained of may be noises in the ear or head, with or without some degree of deafness. This may be due to general causes, such as anæmia, a high or low blood pressure or *neurasthenia*, or to local affections, especially to *otosclerosis*, *chronic otitis media*, bony overgrowth of the external meatus, wax in the ears, labyrinthine lesions or intracranial suppuration. It may occur as an aura in epilepsy, or be produced by drugs, such as quinine and salicylates. The noises may be continuous or intermittent, and of varying character, often hissing or roaring, or at times higher pitched, or pulsating in correspondence with the heart beat. In all cases a complete local examination should be made by an otologist as well as a general examination of the patient by a physician.

*Treatment.* This consists in local measures for relief of Eustachian catarrh, etc., which falls in the province of the specialist, and in addition bromides in doses of gr. 10 to 20 may be given 3 times a day.

*The Vestibular Nerve.* Lesions of the vestibular nerve, or of its terminations in the semicircular canals and otolith organs, result in three main symptoms: Vertigo, postural or kinetic deviation and nystagmus.

*Vertigo.* The patient who complains of vertigo may say that his own body appears to be moving, or that surrounding objects are moving. Vertigo may thus be subjective or objective.

*Etiology.* The common causes of vertigo include: Wax in the ears, otitis media, labyrinthitis, alimentary disturbances, train, car or sea-

sickness, migraine, cerebellar lesions, high blood pressure, over-smoking, alcohol and epilepsy. A cysticercus in the IV ventricle may also produce attacks of severe vertigo. Vestibular vertigo results from irritative lesions of the ear affecting the semicircular canals. Epidemic vertigo, with uncontrollable vomiting, is also described. This may be a manifestation of influenza. Relief may be obtained by ice-cold water-bottles applied to the occipital region, and by the administration of dextrose 1 or 2 id by mouth.

**Postural Deviation** The patient may tend to fall towards the affected side in irritative vestibular lesions, when he stands with eyes closed and feet together.

**Kinetic Deviation** This is described later (see below).

**Nystagmus** Irritative labyrinthine lesions produce spontaneous vestibular nystagmus to the opposite side, *i e*, the eyes make a slow movement to the same side and a rapid twitch back to the opposite side. These movements are increased when the patient looks to the

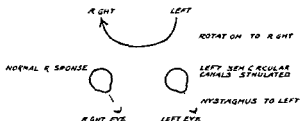


FIG 41 DIAGRAM OF NYSTAGMUS (THE ROTATION TEST)

side of the nystagmus (*i e*, that in which the rapid movement occurs).

**Clinical Findings** Certain tests are applied in suspected cases of labyrinthine lesions. These include 1 *The Rotation Test*. The patient is placed in a chair, with a head rest, which can be rapidly rotated. This is done 20 times. The chair is then stopped, and the patient looks in a direction opposite to that in which he has been rotated. Normally there is nystagmus in a direction opposite to that in which he has been rotated. Thus if he has been rotated from left to right, the rapid nystagmoid movements occur to the left (see Fig 41). The nystagmus persists for about 30 seconds. When the rotation is from left to right, the left labyrinth is chiefly stimulated, and *vice versa*. Feeble nystagmus or absence of nystagmus when the patient is rotated one way or the other, indicates a paralytic lesion of the semicircular canals on the side which is being tested.

2 *The Caloric Test*. The patient lies down, and the external auditory meatus is syringed with cold water (68° F). The drum must first be inspected to see that it is not perforated, and that the external auditory meatus is free from wax or polyp. Cold air may also be used as a stimulus, the air being blown through a coiled metal tube, cooled by a spray of ethyl chloride. If no response is obtained with cold stimuli, hot water (120° F) is sometimes used, but it may upset the patient, causing severe vertigo and vomiting. If the labyrinth is intact, the

normal response to cold is a nystagmus away from the tested side (see Fig. 42); whereas a hot stimulus results in nystagmus towards the tested side. The labyrinth on each side can thus be tested.



FIG. 42. DIAGRAM OF NYSTAGMUS (THE CALORIC TEST).

If the nystagmus is not produced there is a paralytic labyrinthine lesion.

3. Kinetic Deviation. *Bárány's Pointing Test*. The patient sits opposite to the physician, and raises his arm with the elbow straight to touch with his finger the tip of the physician's finger. He then drops his arm to the side and repeats the movement with his eyes closed. If the labyrinth on one side is paralysed he tends to deviate or point past

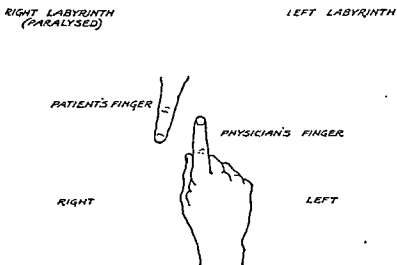


FIG. 43. DIAGRAM OF BÁRÁNY'S POINTING TEST.

to the side of the lesion. If, however, the labyrinth is stimulated, he deviates away from the affected side (see Fig. 43).

**Irritative Vestibular Lesions.** These are exemplified in *Ménière's syndrome* and in *Ménière's symptom complex*. *Ménière's syndrome*. The disease, originally described by Ménière, was due to an acute labyrinthine hæmorrhage associated with acute purulent labyrinthitis, and was not typical of what we now call Ménière's disease. In some cases there is pressure on the auditory nerve caused by an aneurysm of the basilar artery, an abnormally large internal auditory artery, or by a cerebello-pontine angle tumour. It is probable that only lesions affecting the vestibular division of the VIII nerve cause Ménière's

disease. Some authorities believe that it may be caused by faulty water or salt metabolism resulting in a water logged labyrinth. The patient, who is usually an adult, is suddenly attacked with severe vertigo and falls. There may be a preliminary phase of low pitched tinnitus. Vomiting may occur.

*On Examination.* The patient is usually found lying on the sound side, if he attempts to turn on his back or on the affected side, vomiting is induced. The eyes show coarse nystagmoid movements to the affected side. There may be momentary loss of consciousness. Permanent deafness usually ensues. *Meniere's symptom complex.* This may be due to vascular spasm of the internal auditory artery causing alteration in the tension of the endolymph or to otosclerosis. The attacks resemble those of the Ménière's syndrome, but between the attacks there is no loss of hearing.

*Treatment.* Quinine sulphate in doses of gr  $\frac{1}{2}$  t d s may be administered to diminish the sensibility of the labyrinth. In addition aspirin gr 10 t d s may be given, or pot brom gr 20, or Luminal (phenobarbitonum B P) gr  $\frac{1}{2}$  nocte. The most effective medical treatment consists in the administration of a salt poor diet (see p 455) with restriction of the fluid intake to 40 oz in the 24 hours. Ammon chlorid 0.5 G stearetttes 6 t i d with meals, should be given for 3 days out of every five. Salyrgan (mercalylum B P) (see p 228) may also be injected. Favourable results have also been reported by the administration of pot chlorid gr 15, aquam ad m 60, in 60 in water 6 to 8 times a day. The dietary should then have a normal sodium content. Surgical treatment consists in division of the auditory nerve or of the vestibular branch alone. Determination of the side affected is not always easy. The tinnitus and deafness are usually more severe on one side and the caloric tests may indicate which labyrinth is at fault. Vertigo is not always, and tinnitus is rarely, relieved by the operation. Surgery should only be advised if medical treatment fails.

*Acute Vestibulitis.* This is due to acute inflammation of the cochlea and labyrinth. There is tinnitus, vertigo, vomiting and usually pyrexia, and it often results in permanent deafness.

## The IX Nerve

### (The Glossopharyngeal Nerve)

*Anatomy.* The IX nerve is both motor and sensory in function. The motor fibres arise from the nucleus ambiguus in the medulla. This nucleus is common to the IX and X nerves. The nucleus ambiguus extends through the medulla from the level of the VIII nerve above to the decussation of the fillet below. The sensory fibres arise from cells in the superior (jugular) and petrosal ganglia. These ganglia lie on the nerve trunk in the jugular foramen. The sensory fibres pass into the medulla and run down it as the tractus solitarius, the fibres ending at different levels in the nucleus of the tractus solitarius which adjoins the tract (see Fig 14). Other ascending fibres enter the dorsal nucleus and pass up the medulla. The further central course of the sensory

fibres is not definitely known. It will be remembered that the fibres of the pars intermedia of Wrisberg also end in the tractus solitarius (see p. 372). The sensory and motor fibres of the IX nerve enter and leave the medulla in 5 or 6 strands on the postero-lateral surface between the olivary and restiform bodies. The nerve passes through the jugular foramen with the X nerve, but in a special sheath of dura mater. It runs between the internal and external carotid arteries and then deep to the hyoglossus muscle to the pharynx.

*Sensory Branches.* In the skull it receives a tympanic branch (Jacobson's nerve) from the petrosal ganglion, which supplies the middle ear, the mastoid air cells and the Eustachian tube. It conveys taste sensation from the posterior third of the tongue and from taste buds on the soft palate, epiglottis and arytenoid cartilages. It transmits common sensation from the back of the tongue, tonsil, part of the soft palate and the upper part of the pharynx.

*Motor Branches.* The IX nerve supplies motor fibres to the stylopharyngeus, palatopharyngeus and palatoglossus muscles, and secretory fibres to the parotid gland. The constrictors of the pharynx are probably supplied by the vagus.

*Lesions of the IX Nerve.* Nuclear lesions may be due to such causes as a tumour, a gumma, syringomyelia, disseminated sclerosis, polioencephalitis, or thrombosis of the posterior inferior cerebellar artery. There is no definite pathology of central lesions.

*Clinical Findings. Motor Lesions.* It is probable that lower motor neurone lesions causing dysphagia are due to affections of the X nerve. A lesion paralysing the stylopharyngeus muscle may cause some dysphagia, as the larynx is not then drawn up on swallowing and food may enter the trachea.

*Sensory Lesions.* Loss of taste over the posterior third of the tongue, with anaesthesia of the pharynx and tonsillar region and loss of the pharyngeal reflex may result from involvement of the tractus solitarius in the medulla.

*Glossopharyngeal Neuralgia.* The patient is usually an adult over middle age who complains of pain, often agonising, on swallowing or talking, in the region of the pillars of the fauces on one side. The pain may shoot to the ear and disturb sleep. Pressure on the tonsil on the affected side may produce a paroxysm of pain. If the tympanic branch alone is affected, there is deep-seated pain in one ear which occurs in paroxysms, but is not aroused by swallowing.

*Treatment.* There is usually no treatment available for nuclear lesions, unless due to syphilis. Glossopharyngeal neuralgia should be treated first with a gelsemium mixture, as for trigeminal neuralgia (see p. 370). The inhalation, while the patient lies down, of trichlorethylene, 10 t.d.s. may afford relief. This is put up as a sterule encased in cotton-wool and silk. It can be broken between the fingers and the vapour inhaled. If this fails an operation should be considered. The glossopharyngeal nerve may be divided intracranially, or it may be exposed at its emergence from the jugular foramen and avulsed from its central connection with the medulla.

## The X Nerve

*(The Vagus The Pneumogastric Nerve)*

**Anatomy** The vagus nerve contains motor and sensory fibres. The bulbar portion of the spinal accessory nerve is considered to be a part of the vagus nerve. The motor fibres arise from the nucleus ambiguus in the medulla (see Fig 44). The sensory fibres are derived from cells in the trunk ganglion (ganglion nodosum) which lies on the nerve just external to the skull. The sensory fibres enter the medulla and terminate in the tractus solitarius (descending) and in the dorsal nucleus (ascending) (see Fig 44). The upper motor neurones arise

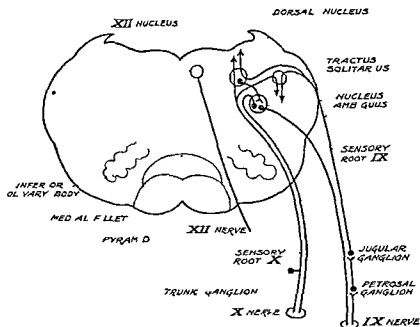


FIG 44 DIAGRAM OF THE IX, X, AND XII CRANIAL NERVE NUCLEI

in the cerebral cortex in the lower part of the precentral gyrus, and pass down in the pyramidal tract to end around the motor nuclei of the opposite side. The vagus nerve emerges from the side of the medulla by a series of roots in line with those of the IX nerve above. A meningeal and an auricular branch leave the nerve at the level of the root ganglion. The latter, Arnold's nerve, supplies the skin at the back of the pinna and the external auditory meatus. The vagus leaves the skull through the jugular foramen behind the IX nerve. Two branches arise from the trunk ganglion, the pharyngeal and the superior laryngeal nerves. The bulbar fibres of the IX nerve join the X nerve just below the trunk ganglion. The vagus runs down the neck in the carotid sheath and gives off the recurrent laryngeal branch and cardiac branches. It enters the thorax, passing between the internal carotid



and subclavian arteries on the left side, and crossing in front of the subclavian artery on the right side. It then traverses the superior and posterior mediastinal spaces. In the thorax it gives rise to the pulmonary and œsophageal plexuses. The vagus enters the abdomen through the œsophageal opening of the diaphragm, the left nerve being in front of, and the right nerve behind the œsophagus. It terminates in branches to the stomach and small intestine. Motor fibres are supplied to the soft palate, the pharynx, larynx, œsophagus, stomach, small intestine, trachea and bronchi. The muscles of the palate, pharynx and larynx are probably supplied by the bulbar portion of the XI nerve. The nerve supply of the laryngeal muscles is described on p. 120.

Secretory fibres are conveyed to the stomach, small intestine and pancreas. Inhibitory fibres are carried to the heart. Sensory impulses pass by the vagus from the skin of the pinna and external auditory meatus, and from the mucous membranes of the larynx, respiratory tract, œsophagus and stomach.

*Lesions of the X Nerve.* *Nuclear Lesions* may be due to progressive bulbar palsy, syringomyelia, encephalitis lethargica, rabies, thrombosis, disseminated sclerosis, or syphilis. *The nerve* may be injured at various sites, such as : (a) *In the skull.* This may be due to basal meningitis, especially syphilitic, a tumour, an aneurysm of the vertebral artery, or a fractured base. (b) *In the neck.* The lesion may be due to a wound, a tumour or to damage at an operation. (c) *In the thorax.* An aneurysm, a mediastinal tumour, or enlarged glands may compress the nerve.

The vagus nerve may be affected by neuritis, especially in diphtheria. Less often the neuritis is due to typhoid fever, influenza, pneumonia, or to chemicals, such as alcohol or arsenic.

*Clinical Findings.* (a) *Irritative Lesions.* Irritation of the vagus in the mediastinum by enlarged glands or a tumour may cause an intractable cough somewhat resembling whooping-cough. Attacks of laryngeal spasm, as in the laryngeal crises of tabes, may be due to central lesions. Reflex laryngeal spasm also occurs in such conditions as laryngismus stridulus (see p. 119). Irritation of the vagus may also cause hyperæsthesia of the posterior wall of the external auditory meatus. Touching the skin in this area may provoke cough (the tragus sign). Some instances of dyspnoea on slight exertion or of bradycardia may be due to vagal stimulation, and asthma is thought to be associated with undue irritability of the broncho-motor portion of the vagus nucleus. Irritation of the pharyngeal fibres of the vagus may account for the dysphagia in hydrophobia or in hysteria. Similarly the gastric crises of tabes may be due to irritation of the gastric branches of the vagus.

(b) *Paralytic Lesions.* Paralysis of the vagus nerve on one side results in anæsthesia and paralysis of the palate and larynx on the same side (the syndrome of Avellis). The palatal paralysis is detected by inspecting the palate when the patient says "ah." The raphe of the palate and the uvula then deviate to the sound side. The oculo-cardiac reflex is also usually abolished on that side. This is tested by pressing

on the eyeball with the eyelid closed. Normally the pulse rate should slow by about 10 to 20 beats a minute. With a vagal paralysis this reflex slowing may be absent. With bilateral lesions affecting the palate, there is usually nasal regurgitation and a nasal voice. With pharyngeal paralysis there is dysphagia and liquids often enter the larynx and provoke cough. The effects of paralysis of the laryngeal branches of the vagus are described on pp. 121, 122.

### The XI Nerve

#### *(The Spinal Accessory Nerve)*

**Anatomy.** The bulbar portion of this nerve has been described above (see p. 382) under the X nerve, of which it constitutes a part. The spinal portion is motor in function, and arises from a column of cells in the lateral part of the anterior horn of grey matter of the spinal cord, from the first to the fifth cervical segments. The nerve fibres emerge between the anterior and posterior nerve roots, and joining to form the XI nerve ascend by the side of the cord in the subdural space to the foramen magnum. It is here joined by the bulbar portion of the nerve, which again shortly leaves it to pass to the vagus nerve just below the trunk ganglion. The XI nerve leaves the skull through the jugular foramen with the X nerve, and passes between the internal carotid artery and the internal jugular vein. It then runs backwards through the sternomastoid muscle leaving its posterior border at the level of the junction of its upper and middle thirds. It finally crosses the posterior triangle of the neck to the under surface of the trapezius muscle. The XI nerve supplies the sternomastoid and part of the trapezius muscles. The trapezius is supplied in its upper part by the XI nerve and to a lesser degree in its lower part the central portion being innervated by the subtrapezial plexus from the 3rd and 4th cervical nerves.

**Lesions of the XI Nerve.** 1 *Nuclear*, as in progressive muscular atrophy, disseminated sclerosis, syringomyelia or myelitis. 2 *At the base of the brain*, the lesion being due to meningitis, aneurysm of the vertebral artery or a fractured base. 3 *In the neck*. Trauma by lifting heavy weights or by a wound. Injury during an operation for glands. Cervical caries. Neuritis is not common.

**Clinical Findings.** Irritative lesions of the XI nerve or of its supranuclear connections may cause spasmodic torticollis. Paralytic lesions. The sternomastoid or trapezius muscles may be paralysed individually or together. With unilateral sternomastoid paralysis the patient does not notice any weakness.

**On Examination.** No deformity is visible but if the patient is asked to turn his head away from the affected side while the examiner's hand is held against his chin on the sound side, the sternomastoid muscle does not contract and stand out on the affected side. If both sternomastoids are paralysed the head is inclined to fall backwards. When the trapezius on one side is paralysed the patient notices difficulty in raising the corresponding arm above the horizontal, in shrugging the shoulder.

or in causing the scapula to approach the mid-line. There is drooping of the shoulder on the affected side. The scapula is in an abnormal position, being farther from the mid-line and lower than normal, and rotated outwards. The vertebral border is prominent and runs from below upwards and outwards; this is known as "winging" of the scapula and is best seen when the arm is held forwards and below the horizontal level. When the patient tries to shrug his shoulders or to approximate the scapulæ, the trapezius muscle can be seen and felt to remain uncontracted on one side. If both trapezii are paralysed the head is inclined to fall forwards. Fibrillary twitchings may be seen in the paralysed muscles which also give a reaction of degeneration.

## The XII Nerve

### (The Hypoglossal Nerve)

**Anatomy.** The nucleus of origin is situated in the medulla, near the floor of the IV ventricle, extending from the level of the stria medullares above to the pyramidal decussation below (see Fig. 44). The nerve fibres leave the medulla between the pyramid and the olive in a series which unite to form three roots. These join and the hypoglossal nerve leaves the skull through the hypoglossal canal (anterior condylar foramen). The nerve passes between the internal carotid artery and the internal jugular vein, and forming connections with the first and second cervical nerves, reaches the floor of the mouth to supply the intrinsic and extrinsic muscles of the tongue.

**Lesions of the XII Nerve.** The lesions may be: 1. *Supranuclear.* These are usually vascular, in the cerebral cortex or internal capsule. 2. *Nuclear.* Various lesions may be found, such as those due to progressive bulbar paralysis, syringobulbia, polioencephalitis, disseminated sclerosis, thrombosis, a gumma or a tumour. 3. *At the base of the brain.* The nerve roots may be affected by a tumour, by meningitis, an aneurysm of the vertebral artery, or a fractured base. 4. *In the hypoglossal canal.* Periostitis may cause compression of the nerve. 5. *In the neck.* Enlarged glands or caries of the first cervical vertebra. Neuritis rarely occurs in association with alcohol or lead.

**Clinical Findings.** 1. *Upper Motor Neurone Lesions.* There is weakness and spasticity of the affected half of the tongue. On protrusion it deviates towards the paralysed side. There is usually hemiplegia of the same side of the body.

2. *Lower Motor Neurone Lesions.* Nuclear lesions are often bilateral, as in progressive bulbar palsy (see p. 394). There is difficulty in chewing and swallowing and in speech. The lips and palate are often affected as well. The lesion may involve one or other pyramidal tract, with hemiplegia. The tongue lies flaccid and shrivelled on the floor of the mouth.

**With Unilateral Lesions.** The affected half of the tongue is small and shrivelled. On protrusion the tongue deviates to the weakened side. Fibrillary contractions may be seen on the affected side of the

tongue, and a reaction of degeneration may be present. Speech is not usually affected.

## SYPHILIS OF THE NERVOUS SYSTEM

**Introductory** Syphilitic lesions of the central nervous system are usually divided into two groups, meningo-vascular (interstitial) and parenchymatous. The former includes such clinical conditions as meningitis, meningo-encephalitis, meningo-myelitis, cerebral and spinal gumma, cerebral syphilis and spinal syphilis, and the latter group embraces general paralysis of the insane and tabes dorsalis. The distinction is, however, somewhat artificial, as the primary cerebral lesion in either case is invariably an arteritis or a lymphangitis. Thus the meninges and vessels are affected predominantly in meningo-vascular syphilis, but in general paralysis of the insane and in tabes lesions can be demonstrated at autopsy in the vessels and meninges, and during life the cerebro-spinal fluid shows a meningeal reaction (increase of cells, etc.).

The nervous system is believed to be infected during the primary or secondary stages of syphilis in cases which subsequently develop neuro-syphilis, although not every case so infected later shows signs of neuro-syphilis. Actually there is evidence of infection of the nervous system in a large proportion of all cases of syphilis during the primary and secondary stages. Thus there may be headache, giddiness and some disturbance of vision. The cerebro-spinal fluid shows an increase of cells, up to 100 per c mm., globulin may be present, and spirochaetes found in the fluid. The Wassermann reaction is also at times positive in the fluid before it is so in the blood, and a luetic curve is obtained with Lange's test (see Fig. 25). In the majority of cases these changes are transitory, but the treponemes may lie dormant and after 4 or 5 years another meningeal reaction occur, with clinical evidence of neuro-syphilis.

### Meningo-vascular Syphilis

The lesions may affect chiefly the brain or the spinal cord.

#### Cerebral Syphilis

**Pathology** The meninges may be chiefly affected, the pia arachnoid being involved in a gummatous infiltration. The base of the brain is a favourite site, and various cranial nerves may be affected. In other cases the lesion is cortical. Small or large gummata form and compress the underlying brain substance. The vascular changes consist of endarteritis obliterans, whereby the blood supply is cut off from portions of the brain with consequent softening, hæmorrhages or the formation of cysts.

**Clinical Findings** The patient may give a history of syphilitic infection 4 or 5 years previously. He complains of headache, which is often worse at night, the memory deteriorates, and transient pareses of the limbs or double vision may occur.

In cortical meningitis there may be epileptiform convulsions,

affecting one or more limbs, and during the attacks the patient may or may not lose consciousness.

In *basal meningitis* various cranial nerve lesions can be detected, such as optic neuritis, unequal, irregular or fixed pupils, ptosis, weakness of the external ocular muscles, pains in the face, deafness, weakness of one side of the tongue, or unilateral vocal cord paralysis.

A *cerebral gumma* gives rise to the signs of a cerebral tumour, there is optic neuritis, and often vomiting, with headache.

*Syphilitic dementia* may occur in meningo-vascular syphilis, the symptoms closely resemble those of general paralysis, but there is usually evidence of some cranial nerve lesion.

### Spinal Syphilis

**Pathology.** The dura may be much thickened, especially in the cervical region, giving rise to *pachymeningitis cervicalis hypertrophica*. There is usually compression of the cervical nerve roots. In other cases there is a gummatous infiltration of the pia-arachnoid; especially in the thoracic region. The pia becomes adherent to the cord and the vascular supply of the cord is interfered with. A condition of meningo-myelitis then results. Thrombosis or a hæmorrhage in the cord will give rise to acute syphilitic myelitis. A gumma may also form, with symptoms of a cord tumour.

**Clinical Findings.** *Pachymeningitis Cervicalis Hypertrophica.* The patient complains of severe pains in the neck and upper part of the back and arms, with weakness of the arm muscles.

**On Examination:** There is wasting of the arm muscles, with diminution of the deep reflexes and some loss of cutaneous sensation.

**Chronic Meningo-myelitis.** The patient complains of pain in the back, usually in the thoracic region, with weakness and numbness of the legs and often loss of sphincter control.

**On Examination:** Signs of a lesion of the cord in the thoracic region are found. Thus there is weakness of the legs or paraplegia, with spasticity of the leg muscles and increase of the deep reflexes. The plantar responses are extensor. The abdominal reflexes are lost below the site of the lesion. There is some diminution of sensation or anæsthesia over the legs and lower half of the body below the lesion. The disease runs a course usually lasting a few months, when, with suitable treatment, recovery may gradually take place.

**Erb's Syphilitic Paralysis.** This usually develops insidiously several years after infection; there is spasticity of the legs, without marked changes of sensation.

**Acute Transverse Myelitis.** The patient is suddenly taken ill with weakness or paralysis of the legs and loss of sphincter control. The signs closely resemble those described above for meningo-myelitis, but the paralysis is usually of a flaccid type in the early stages, with loss of deep reflexes and of the plantar response. Later a spastic paralysis may ensue with exaggerated tendon reflexes and extensor plantar responses. A zone of hyperæsthesia is usually found running round the body at the skin level corresponding with the site of the lesion in the cord. In

both these varieties there is often evidence of some intra cranial involvement such as headache, giddiness and weakness of the external ocular muscles

**Differential Diagnosis** The effects produced by meningo vascular syphilis may and do often closely simulate many diseases of the nervous system such as epilepsy, Jacksonian epilepsy, a cerebral tumour, meningitis due to other causes general paralysis, tabo paresis, disseminated sclerosis, progressive muscular atrophy, etc The presence of cranial nerve palsies is always suggestive of a syphilitic infection The diagnosis is established by the results of the examination of the blood and of the cerebro spinal fluid. The blood Wassermann reaction is nearly always positive and the cerebro spinal fluid shows a positive Wassermann reaction increase of cells and presence of globulin and often a meningitic curve (see Fig 25)

**Prognosis** This is most hopeful when the lesions are meningeal rather than vascular

**Treatment.** Mercury should first be administered, best byunction of blue ointment (ung hydrarg) gr 60 3 days a week until a course of 60 inunctions has been given A careful watch must be kept on the gums for signs of mercurialism Pot iod gr 5 to 40 t d s is also given simultaneously by mouth A course of 6 injections of neoarsphenamine is then given intravenously beginning with 0.1 G, then 0.3 G 0.45 G, and working up to 0.6 G or 0.9 G Subsequently Hutchinson's pill (hyd c creta gr 1 pulv ipecac et opii gr 1) is given daily for 2 years with courses of a month's duration 3 times a year afterwards for several years The neoarsphenamine course should be repeated after a 6 months' interval

### Parenchymatous Syphilis

*This includes tabes dorsalis and general paralysis of the insane*

#### Tabes Dorsalis

*(Locomotor Ataxia)*

**Definition** A disease characterised by pains, alteration of sensation, hypotonus, inco ordination, loss of deep reflexes, trophic and visceral changes

**Etiology** Tabes dorsalis is caused by infection with the *Treponema pallidum* The infection is generally acquired in adult life in juvenile tabes the disease is congenital or rarely due to an accidental infection of the child Only about 1 to 5% of those infected with syphilis develop tabes **Predisposing causes** 1 Age Between 20 and 50 years 2 Sex Males predominate except in juvenile tabes An injury appears in some cases to light up the disease

**Pathology** In the majority of cases of syphilis there is infection of the central nervous system during the secondary stage. The infection may die out or lie dormant, and after several years the treponemes may give rise to lesions of the posterior nerve roots of the spinal cord, usually in the lumbo sacral region The lesion is essentially a fibroblastic infiltration of the sheath of the posterior nerve roots, which

spreads into the nerve, and in which treponemes are demonstrable. The exogenous nerve fibres, whose cells of origin are situated in the posterior root ganglia, are thus affected and degenerate. The fibres conveying sensations of pain, touch and temperature end in the substantia gelatinosa Rolandi on entering the cord, and a fresh relay carries the impulses up in the spino-thalamic tracts. The latter tracts do not degenerate (except in advanced cases), as they are composed of endogenous fibres, although their impulses are affected owing to interference with the exogenous fibres in the posterior roots. Sclerosis of the posterior columns results from the overgrowth of the neuroglial tissue. In cervical tabes the changes are most marked in the upper part of the cord. The I and II cranial nerves may be affected by a perivascular infiltration with lymphocytes and plasma cells. Less frequently the V, VIII, X and XII nerves are involved by a fibroblastic infiltration. Degeneration of the colliculo-nuclear fibres in the mid-brain connecting the higher visual centres in the superior corpora quadrigemina with the III nucleus is thought to give rise to the Argyll-Robertson pupils (see Fig. 32). The bones may be rarefied, and the joints show atrophic or hypertrophic bony changes with fluid formation in the synovial membrane (Charcot's joints, see below). In some cases the anterior roots of the cord show degenerative changes.

**Clinical Findings.** The patient is usually an adult male, who may, or may not give a history of syphilitic infection some years previously. He may complain first of an abnormality in micturition, such as delay in beginning, or of frequency or incontinence. Impotence may also be an early symptom. In other cases pains have been noticed, either prolonged muscular ones, which are probably described as rheumatic, or sharp agonising "lightning" pains, occurring in attacks of sharp stabs. These are often felt near the outer side of the knee, in the calf, heel or foot, or the pains may run up and down the leg. In other cases the first symptom is dimness of vision or transient diplopia, or there may be complaints of difficulty in walking, the foot appearing to catch in the ground, or unsteadiness may be noted, especially in the dark or when the eyes are closed as in washing the face. Numbness, tingling, or sensations of cold may be felt in the legs or trunk, or a sense of constriction or pain around the trunk, often at the level of the upper part of the abdomen (girdle sensation). Various tabetic crises may cause distress. Very acute pain may occur in the epigastrium, with nausea, vomiting and faintness or even hæmatemesis (gastric crises). Intestinal crises are characterised by attacks of diarrhoea, rectal crises by tenesmus, vesical and renal crises by suprapubic pain and frequency, urethral crises by pain in the urethra, nasal crises by sneezing, and laryngeal or bronchial crises by dyspnoea and cough. Præcordial pain may occur with cardiac crises.

**On Examination :** The physical signs found in an early case are very variable and few cases pass through clearly defined pre-ataxic, ataxic and paralytic stages. The ankle-jerks are lost early. Areas of altered cutaneous sensation may be detected, such as patches of anæsthesia to the pin prick, light touch or temperature, especially over the tibiae, the

perineum, the inner side of the arms or on the tip of the nose. There may be definite delay in appreciating painful stimuli. Hyperæsthesia to light touch may be present, especially over the trunk. The bone vibration sense may be lost in the feet or legs. The pupils may show the Argyll Robertson sign, being small and the reaction to light being lost while that to accommodation persists. The pupils may be irregular, unequal and pin point in size. Ataxia may be demonstrated by asking the patient to stand with his feet and toes together and eyes shut. The patient may sway or fall (Romberg's sign) and should be carefully watched during the test. In a less advanced stage he is unable to stand on one foot with his eyes shut or to walk along a line with his eyes open, placing one foot in front of the other. In cervical tabes the arms are chiefly affected and the patient cannot touch the tip of his nose with one finger, with his eyes closed. Pressure over the tendo Achillis (Abadie's sign) or over the ulnar nerve (Biernacki's sign) may not cause pain. Optic atrophy may be an early sign, the discs being pearly white. The external rectus muscles may be weak so that there is strabismus or diplopia, and weakness of the levator palpebre superioris may result in ptosis. The tone of the leg muscles may be much diminished and the joint ligaments lax so that the knee can be hyperextended and the hip hyperflexed. Trophic changes, such as a perforating ulcer on the sole of the foot, or painless swellings of joints, may occur. The latter constitute Charcot's arthropathy. Large joints, such as the knee, hip, ankle, elbow or shoulder, are usually affected. The X ray of the joints may show atrophy or some hypertrophy of bone. Later the patient may become very ataxic, standing on a wide base and requiring two sticks to walk, and often throwing up the legs and bringing them down in a stamping manner, heel first. Still later he may become paralysed and bed ridden. The blood. The Wassermann reaction is positive in about 70% of early cases. The cerebro spinal fluid. Changes are found early in the disease and are of great diagnostic importance. The Wassermann reaction is positive in about 70 to 90% of early cases. The colloidal gold curve (Lange's test) gives a luetic response (see Fig 25) in about 85% of cases. This is not diagnostic of tabes, but differs from the parietic curve. The cells are increased to about 80 per c.mm., there being a pleocytosis in a 100% of cases. Globulin is present (Nonne-Apelt test) in about 90% of cases.

**Differential Diagnosis.** As the most hopeful results are to be expected if treatment is applied early, the diagnosis should not be delayed until the disease is firmly established as shown by absent knee-jerks, an Argyll Robertson pupil and marked ataxia. In the early stages peripheral neuritis due to diabetes or alcohol may be mistaken for tabes. The urine should be tested for sugar in every case. The pains in the limbs may be considered to be rheumatic. The gastric crises closely simulate an acute abdominal lesion, and if other signs of tabes are not looked for, an unnecessary operation may be performed. A condition resembling the Argyll Robertson pupil may also be met with in encephalitis lethargica, and in alcoholic neuritis, but the pupils here are often neither small nor irregular and the reaction to



accommodation is also defective. The myotonic pupil is nearly always unilateral and larger than its fellow, it fails to react to light unless the patient is kept for some time in a dark room. The reaction to convergence is prolonged and sometimes there is no reaction to accommodation. It may be met with in young women who are apparently in good health and often the knee and ankle-jerks are absent. Difficulty in walking may occur in disseminated sclerosis, but here the plantar response is extensor and this also applies to Friedreich's ataxia. In cerebellar ataxia there is usually nystagmus, and the ataxy is not increased when the eyes are shut. Syphilitic meningo-myelitis may closely simulate tabes, but the course is usually more acute.

**Course and Complications.** The course is very variable, and the disease may be arrested at any stage, whereas an acute intercurrent illness may accelerate its progress. In the elderly it tends to run a benign course. Optic atrophy usually means that the patient will be blind in a few years. Complications include cystitis and pyelonephritis. Dislocation of the hip may result from atrophy of the head of the femur. General paralysis of the insane sometimes occurs as a complication.

**Prognosis.** This is very variable. Death may rapidly ensue, the patient wasting and becoming bedridden, or developing some complication, such as an intercurrent disease or infection of the urinary tract. In other cases the disease is arrested for long periods.

**Treatment.** A preliminary period of rest in bed for a few weeks is always of value. During this time a course of 10 injections of neoarsphenamine should be given, beginning with an intravenous injection of 0.1 G. and increasing the dosage gradually to 0.6 or 0.9 G. at weekly intervals. Subsequently the course is repeated in 3 months' time. During the interval mercury is administered by inunction, ung. hydrarg. gr. 60 is rubbed into the skin 3 days a week until 50 or 60 inunctions have been given. A careful watch must be kept on the gums for signs of mercurialism. The malaria treatment (see p. 393) has been used for tabes. The results are not so satisfactory as in the case of general paralysis of the insane, and it is only of value in the active inflammatory stage of the disease. Ataxia can be improved by having the soles of the shoes weighted, and the patient can be trained to regulate his movements by means of his eyes, by Fraenkel's exercises. The patient practises placing his feet in certain positions and on certain marks, both when he is lying and on standing. When the disease is arrested or "burned out" the cerebro-spinal fluid usually shows an absence of globulin, and no colloidal gold curve, and the cell count is low; the Wassermann reaction is usually negative. Gastric crises may be treated by drop doses of liq. iodi mit., m. 1 to 2 in fl. oz. 1 water every hour, or by Chlorotone (chlorbutol B.P.) gr. 10 in a cachet every 4 hours. If relief is not obtained an intravenous injection of 5 mls of calcium gluconate (B.P.Add.) or of atropin. sulph. gr. 1/100 may prove successful. Laryngeal crises can generally be relieved by an inhalation of m. 5 of amyl nitrite. For the "lightning" pains phenazone gr. 10 may be

given. Care should always be taken to see that no residual urine is left in the bladder. If so, it should be removed daily by catheter. If the urine is all alkali it is advisable to give acid sodium phosphate gr 60 at night to render it acid. When there is hesitancy in micturition, the nuc vom m 10 to 15 t d s is of value. A Charcot's joint should be kept at rest in a splint.

## General Paralysis of the Insane

### (*Dementia Paralytica*)

**Definition** A disease characterised by progressive deterioration of the mind with paralysis.

**Etiology** *Dementia paralytica* is caused by infection with the *Treponema pallidum*. The infection is usually acquired but congenital syphilis may give rise to juvenile general paralysis. *Predisposing causes* 1 Age Usually between 30 and 50 years. 2 Sex Males predominate. Mental worry or brain work may also predispose.

**Pathology** The body generally is wasted at autopsy. The skull cap is thickened. The dura mater is dense and hæmorrhagic pachymeningitis may be present. The arachnoid is thickened and the pia mater is adherent at places to the brain so that on stripping it off, small portions of brain are removed with it. The brain is atrophied, the left hemisphere being more affected than the right in right handed people. The convolutions are flat and the sulci wide but shallow. The ventricles are dilated and the ependyma lining the floor of the fourth ventricle is granular and rough (*langue du chat*). The amount of cerebro spinal fluid is increased. The grey matter is seen on section to be diminished. Microscopically treponemes may be found, especially in the frontal poles. The cortical pyramidal cells are degenerated. There is a cortical perivascular infiltration with lymphocytes and plasma cells, especially in the frontal lobes and to a lesser degree in the mid brain and cerebellum. The neuroglial cells are proliferated. Syphilitic lesions in other parts of the body are not often found.

**Clinical Findings** A history of syphilitic infection about 10 or 15 years previously may or may not be obtainable. In the early stages the patient may complain of headache or of a sensation of oppression on the top of the head, with insomnia. Those who know him best will first detect alteration in his character, such as lack of attention to details, failing memory, passion, emotion, deterioration of judgment and intellect, carelessness in habits or in dress and possibly laxity of morals. Various delusions may occur, such as those of grandeur, wealth, excessive health, accomplishments, or marked depression. Epileptiform convulsions may occur or congestive attacks of an apoplectic type followed by hemiplegia or monoplegia, which gradually pass off.

**On Examination** The pupils are usually unequal and irregular, and they often show the Argyll Robertson condition reacting to accommodation but not to light. Tremors are seen in the lips and tongue. The tone of the facial muscles diminishes, so that wrinkles

disappear. Tremors may also be seen in the hands. The speech is altered so that it is hesitating or slurred, and consonants such as the linguals and dentals are indistinct. If the patient is asked to write, the words are shaky and certain syllables are omitted. Gradually the gait becomes unsteady and the legs are weak. The tendon reflexes are exaggerated in the early stages, but later the knee-jerks and ankle-jerks may be lost. The plantar response is usually flexor, but later it may become extensor. At this stage there is a spastic paresis of the legs and the sphincter control of the bladder and rectum may be lost. In the terminal stage the patient is bed-ridden and trophic changes, such as bedsores, are likely to develop. Some degree of optic atrophy is not uncommon. The blood: The Wassermann reaction is positive in nearly 100% of cases. The cerebro-spinal fluid: The fluid is clear. The pressure is slightly increased. The cells are increased to about 50 to 200 lymphocytes per c.mm. Large mononuclears or plasma cells may also be present. The Nonne-Apelt test for globulin is positive. The Lange colloidal gold test gives a typical paretic curve (see Fig. 25). The Wassermann reaction is positive in about 95 to 100% of cases.

**Differential Diagnosis.** In the early stages general paralysis may be mistaken for a functional disorder of the nervous system, such as neurasthenia. Cerebral syphilis is characterised by involvement of certain of the cranial motor nerves. In chronic alcoholic dementia the pupils may not react to light and there are marked tremors, but the examination of the cerebro-spinal fluid serves to differentiate. A frontal lobe tumour of the brain may also cause difficulty in diagnosis, but usually signs of increased intracranial pressure develop, such as optic neuritis or vomiting, and the cerebro-spinal fluid does not show the changes characteristic of general paralysis.

**Course and Complications.** The course in an untreated case is progressive, but remissions may occur. In some instances signs of tabes also develop, the disease then being known as tabo-paresis.

**Prognosis.** If untreated, death usually takes place within 4 years of diagnosis. If the disease is arrested by treatment in the early stages, the patient may be restored to health so that he is able to resume his work. If arrest occurs in the later stages, the patient closely resembles an animal with a very low grade intellect; speech is difficult to understand, he is childish and very emotional and requires constant attention.

**Treatment.** The most successful treatment consists in infecting the patient with benign tertian malaria. This can be done either by the bite of an infected mosquito, or by the intramuscular injection between the scapulae of 5 mils of citrated blood taken from a patient suffering from malaria. After about 7 to 10 days, if the infection takes, the patient develops an attack of malaria. He is allowed to suffer about 8 such attacks, and quinine is then given to abort the course of the malaria. Subsequently a course of 6 injections of neoarsphenamine is given, beginning with an intravenous injection of 0.1 G., and at weekly intervals increasing the dose to 0.6 or 0.9 G. Tryparsamide may be used as an alternative to neoarsphenamine. Eight or ten weekly intravenous injections of 3 G. are given.

## DISEASES OF THE MOTOR NEURONES

## Chronic Bulbar and Spinal Atrophic Paralysis

**Introductory** A group of diseases is included under this heading, such as Progressive ophthalmoplegia, progressive bulbar paralysis, progressive muscular atrophy, amyotrophic lateral sclerosis, and primary lateral sclerosis. In all of them the lesion consists of a chronic degeneration of the cells of the lower motor neurones situated in the pons, medulla or cord with changes of varying degree in the upper motor neurones (pyramidal tracts). There is an atrophic paralysis, the degree of spasticity present depending upon the extent of the lesion in the upper motor neurones. This is most marked in amyotrophic lateral sclerosis and in primary lateral sclerosis.

*Progressive Ophthalmoplegia*

**Definition** A disease characterised by paralysis of the external oculo motor muscles, due to degeneration of the nuclei of their lower motor neurones.

**Etiology** The disease is often associated with syphilis. It may occur in tabes dorsalis, or in general paralysis of the insane, or as a manifestation of progressive muscular atrophy.

**Pathology** Degeneration occurs in the nuclei of the III, IV and VI cranial nerves.

**Clinical Findings** The disease usually has an insidious onset, the patient complaining of diplopia.

**On Examination** There is often bilateral ptosis, and weakness or paralysis of various external ocular muscles is found. Later other bulbar nuclei are usually affected, death occurring as in progressive bulbar paralysis (see p. 395).

*Progressive Bulbar Paralysis**(Labio-glosso-pharyngeal Paralysis)*

**Definition** A disease characterised by wasting of the muscles of the tongue, lips, palate and pharynx, due to degeneration of the cells of origin of the lower motor neurones which supply them.

**Etiology** The cause is unknown. *Predisposing causes* 1. Age. Usually over 50. 2. Sex. Males predominate slightly.

**Pathology** There is atrophy of motor cells of certain cranial nerves, especially the X, XI and XII, and less often the V, VII and IX. There is usually some degeneration of the pyramidal tracts. The wasting is seen in the muscles of the tongue, lips, palate, and in the muscles connected with the hyoid bone and the mandible. The pharyngeal, laryngeal and œsophageal muscles are only slightly affected.

**Clinical Findings** The patient first experiences difficulty in speech, especially in pronouncing consonants, such as linguals and labials, later, disturbance of swallowing, such as nasal regurgitation, may occur, or if the epiglottis is affected, swallowing will provoke coughing attacks. Mastication may also be affected.

**On Examination** When the disease is developed the sufferer from

chronic bulbar paralysis is a pitiable object ; the lower jaw hangs down, saliva dribbles from the mouth, and speech consists of an indistinct mumble. The upper part of the face offers a surprising contrast, being practically unaffected. *The cranial nerves* : I, II, III, IV, VI and VIII are normal. V motor, there is weakness of the muscles of mastication and of those attached to the hyoid bone. V sensory, normal. VII, weakness and wasting of the lip muscles ; the patient cannot whistle. The orbicularis palpebrarum is only slightly affected. IX, X and XI (accessory part), weakness of the palate is present, and the vocal cord adductors may be affected. XII, wasting and paralysis of the intrinsic muscles of the tongue. The tongue is shrivelled and wrinkled and lies paralysed on the floor of the mouth. Fibrillary twitching is marked in the early stages. If the pyramidal tracts are also involved, the knee-jerks are exaggerated. The affected muscles are atonic, there is no jaw-jerk, and the reaction of degeneration is present.

*Varieties.* A tonic atrophic variety of bulbar paralysis is described, in which there is a combination of an upper and lower motor neurone lesion. The tongue is paralysed, but is smaller and firmer than normal. A jaw-jerk is present.

*Spastic Bulbar Paralysis* (pseudo-bulbar paralysis). This results from a bilateral lesion of the upper motor neurones in the brain, usually in the internal capsule. There is often a history of a previous attack of hemiplegia. The paralysed muscles are spastic ; the tongue is smooth, small and firm. There is no reaction of degeneration, and a jaw-jerk is present.

*Differential Diagnosis.* Tumours, vascular lesions or poli-encephalitis of the mid-brain may give rise to a rapidly developing bulbar palsy. There are often sensory changes and the pyramidal tracts may be involved, causing spastic lesions of the arms or legs. In myasthenia gravis there is no atrophy of muscles, ptosis is frequently present, and the muscles give the myasthenic reaction (see p. 603). Diphtheria may cause a transitory bulbar paralysis. Basal syphilitic meningitis or a fusiform atheromatous dilatation of one vertebral artery may result in an unilateral bulbar palsy.

*Course and Complications.* The course is steadily progressive. Complications include inhalation bronchopneumonia, or cardiac and respiratory failure.

*Prognosis.* Death usually occurs within two years from the onset.

*Treatment.* No cure is known. Care should be taken in feeding, semi-solids are most suitable, but in some cases resort must be made to nasal feeding.

### Progressive Muscular Atrophy

#### (*Progressive Spinal Muscular Atrophy*)

*Definition.* A disease characterised by wasting of spinal muscles, due to degeneration of the cells of origin of their lower motor neurones.

*Etiology.* The cause is not known. In some cases there is evidence of syphilis, as shown by a positive blood Wassermann reaction. *Predisposing causes* : 1. Age : Usually between 25 and 40 years, but

it may begin considerably earlier, as at 12 years, or later, as at over 70 years 2. Sex Males predominate.

**Pathology.**—In the majority of cases the lesion is situated in the cervical region of the cord, but any level may be affected. The cord lesion is a degeneration of the anterior horn cells, with or without degeneration of the extramedullary anterior nerve roots. In addition, there is always some evidence pathologically of degeneration of the pyramidal tracts, and of the afferent spino cerebellar tracts arising in Clarke's column (Gowers' and Flechsig's tracts). The muscles affected show wasting of the fibres, healthy fibres often lying side by side with those which have degenerated.

**Clinical Findings.** In the typical cervical type the patient notices the gradual onset of weakness and wasting of muscles, first in one hand, usually the right, and later in the other. There may be aching or numbness in the hand.

**On Examination** There is wasting of the small muscles of the hand, those of the thenar and hypothenar eminences, the interossei and the lumbricals. The hand assumes a claw shaped deformity, with hyperextension at the metacarpo-phalangeal joints and flexion at the interphalangeal joints. The thumb may rotate outwards to lie in the plane of the fingers. Wasting spreads to the flexors of the forearms and then to the scapulo-humeral muscles, such as the deltoid and serratus magnus. Certain muscles are typically spared, such as the lower part of the pectoralis major, the triceps, the latissimus dorsi and the upper part of the trapezius. The neck muscles and intercostal muscles may be paralysed later. Fibrillary contractions are seen early in muscles which are doomed to atrophy. The deep reflexes connected with the affected muscles are abolished. The electrical reactions show a progressive lack of response both to faradisation and to galvanism, as the fibres degenerate, but often there is no true reaction of degeneration. No sensory changes can be elicited. Exaggerated knee-jerks are an indication of a partial involvement of the pyramidal tracts.

Other types of progressive muscular atrophy occur. Thus in the shoulder type, the scapulo humeral muscles are first affected, and changes develop later in the hands. In the neck type there is weakness first of the muscles which support the head, so that it drops forward. In the peroneal type there is weakness of the anterior tibial and peroneal muscles.

**Differential Diagnosis** Other causes of wasting of the small muscles of the hand require consideration, such as peripheral neuritis, a cervical rib, cervical pachymeningitis, cervical caries, syringomyelia and rheumatoid arthritis of the wrist. The absence of sensory changes and the presence of fibrillary contractions in progressive muscular atrophy are of great diagnostic value. The shoulder type is differentiated from the facio scapulo-humeral myopathy (see p. 600) by the muscle groups affected, and by the absence of fibrillary contractions in the myopathy. The peroneal type is distinguished from peroneal muscular atrophy (Charcot Marie Tooth) (see p. 420) by the fact that the latter occurs in children and there is often a familial incidence,

and there may be some sensory changes. It is also diagnosed from the distal type of myopathy (see p. 600) by the fibrillary contractions.

**Course and Complications.** If the onset is acute, then the course is likely to be rapidly progressive. In an average case the disease persists for 5 to 15 years. Complications include the development of signs of amyotrophic lateral sclerosis or of chronic bulbar paralysis.

**Prognosis.** Death occurs in a variable time, as described above.

**Treatment.** There is no cure known.

### Amyotrophic Lateral Sclerosis

**Definition.** A chronic disease of the brain and spinal cord, with degeneration of the upper and lower motor neurones.

**Etiology.** The cause is unknown. *Predisposing causes:* 1. Age: Usually over 40 years. 2. Sex: Males predominate slightly.

**Pathology.** The upper motor neurone tracts in the cord degenerate, the lesion appearing to extend upwards so that the motor cortical cells are affected. The anterior horn cells in the cord degenerate, and certain cranial nerve nuclei may be similarly affected.

**Clinical Findings.** The disease usually starts insidiously with pains or numbness in the fingers, hands or arms. The patient then notices weakness in the hands and arms, with perhaps some stiffness in the legs.

**On Examination:** In an established case there is wasting of the small muscles of the hands with deformity due to contractures (griffin's paw). The muscles of the forearms, arms and shoulders may also be wasted. There is weakness in the affected muscles and the neck may be involved, so that there is difficulty in supporting the head. The muscles show fibrillary twitching, which can also be elicited by tapping over them. Sensation is unaffected. The tendon reflexes in the arms are increased. There is spasticity in the legs with hypertonus, but the power is retained and there is no wasting. Later, walking may become impossible. The knee-jerks and ankle-jerks are increased, there is ankle and patella clonus, and the plantar response is extensor. The abdominal reflexes are increased in the early stages, but later may be lost. The muscles of the arms show a gradual failure of response, both to faradic and galvanic stimulation. Sensation is not affected; the sphincter control is normal. A jaw-jerk is often present, due to degeneration of the upper motor neurone fibres connecting with the motor nuclei controlling the jaw muscles (V motor). Affection of the cranial nerve nuclei may be shown by disturbance of speech (dysarthria), wasting of the facial muscles (orbicularis oris), difficulty in mastication and swallowing, tremors and wasting of the tongue.

**Differential Diagnosis.** If in the early stages the hands or arms alone are involved the case closely simulates one of progressive muscular atrophy, to which the disease is allied, and from which its separation is somewhat artificial, as described on p. 396. Other causes of wasting of the small hand muscles must then be considered (see p. 396). When the legs are primarily affected, the disease resembles primary lateral sclerosis, with which it is also closely connected, from the pathological standpoint. If the cranial motor nuclei are first involved, the case

presents the aspect of bulbar paralysis. In a fully developed case there is little difficulty in establishing the diagnosis.

**Course and Complications** The course may be rapid or comparatively slow. Involvement of the cranial motor nuclei is a serious sign. Death often occurs from inhalation bronchopneumonia, due to interference with deglutition.

**Prognosis** The disease is usually fatal within 1 year from its onset.

**Treatment** This is usually only palliative. In a few cases good results have been reported by the administration of vitamin E (pure synthetic  $\alpha$  tocopherol) in the form of Ephynal tablets, mg 32 to 320. Hot baths, massage and passive movements tend to alleviate the spasticity.

### Primary Lateral Sclerosis

#### (*Lrb s Spastic Spinal Sclerosis*)

**Definition** A disease characterised by spastic weakness of the legs due to degeneration of the pyramidal tracts.

**Etiology** The cause is unknown.

**Pathology** The degeneration of the pyramidal tracts appears to begin in the lower part of the cord.

**Clinical Findings** The patient is usually an adult who notices gradually increasing weakness and stiffness in one and later in both legs.

**On Examination** The typical signs of an upper motor neurone lesion are found. The legs are weak, the tone of the muscles being increased but there is little wasting. Sensation is normal the deep reflexes are increased and the plantar response is extensor. The gait may be of the 'scissor' type, due to spasm of the adductors.

**Differential Diagnosis** The existence of a primary lateral sclerosis as a separate disease is doubted by many authorities. The pyramidal tracts may be similarly affected in disseminated sclerosis, syphilitic sclerosis and amyotrophic lateral sclerosis. A cord tumour or haematomyelia may also give rise to similar signs. Spinal arteriosclerosis may occur in the aged giving rise to paraplegic weakness and stiffness and often there is local muscular atrophy in the arms and hands. In all cases a careful search should be made for signs of disease in other parts of the nervous system and it is only by watching the case for some time that other diseases, such as disseminated sclerosis, can be excluded.

**Course and Complications** The course is slowly progressive.

**Prognosis** The patient may live for 20 to 30 years from the onset of the symptoms.

**Treatment** There is no curative treatment known.

### THE SPINAL CORD

#### Hæmorrhagic

#### (*Meningeal Hæmorrhage*)

**Definition** Hæmorrhage into the meninges of the spinal cord.

**Etiology** The hæmorrhage may be extradural or intradural.



Extradural hæmorrhage results from trauma or from rupture of an aortic aneurysm. Intradural hæmorrhage (spinal subarachnoid hæmorrhage) may be due also to trauma or to rupture of a basilar or vertebral aneurysm. In some cases it is due to convulsions, asphyxia, purpura, hæmophilia, or to hæmorrhagic small-pox. It is a condition which is seldom encountered.

**Pathology.** The cervical region of the cord is most often affected. In some cases there is compression of the cord.

**Clinical Findings.** The onset is usually sudden with severe pains in the back and symptoms of shock.

**On Examination:** There may be rigidity of the back and spasmodic muscular contractions in the arms or legs. A zone of hyperæsthesia, corresponding with the segmental level of cord affected, is sometimes found on the trunk. The temperature is not raised. Blood may be present in the cerebro-spinal fluid on lumbar puncture.

**Differential Diagnosis.** Spinal meningeal hæmorrhage is characterised by the sudden onset and severe root pains. In hæmatomyelia (see below) the pains are not so severe and muscular spasms are not so likely to occur. In meningitis the temperature is raised.

**Course and Complications.** The course is usually rapidly fatal, but in some cases there is recovery with persistent weakness of the limbs. Bed sores, cystitis or pyelonephritis may develop in prolonged cases.

**Prognosis.** Death often occurs in a day or so.

**Treatment.** Laminectomy should be performed to relieve pressure symptoms. In other cases an ice bag may be applied to the spine, and pain assuaged by the subcutaneous injection of morphin. sulph. gr.  $\frac{1}{2}$  to  $\frac{1}{4}$ .

### Hæmatomyelia

**Definition.** Hæmorrhage into the spinal cord.

**Etiology.** Primary and secondary cases are described. *Primary hæmatomyelia:* This may occur suddenly without any apparent cause, or follow an injury to the spine, often of a slight nature, or result from the strain of coughing, sneezing or lifting a weight. At times it results from diving with the neck flexed. *Secondary hæmatomyelia:* This may be due to blood diseases such as purpura, to myelitis, poliomyelitis, syringomyelia, or a tumour of the cord. Minute hæmorrhages are found in the cord in asphyxia, tetanus, convulsions, etc. *Predisposing causes:* 1. Age: Usually between 15 and 30 years. 2. Sex: The lesion is more common in men.

**Pathology.** The hæmorrhage often occurs in the cervical region of the cord; the blood tends to collect in the grey matter, especially the posterior commissure, and in the anterior horn cells. There is œdema around the hæmorrhagic area. The hæmorrhage may extend up and down the cord for a few segments in a spindle-shaped area. It is absorbed, if the patient survives, leaving either a cyst or a scar. The

hæmorrhage thus interferes especially with the sensory tracts conveying pain and temperature sensations, with the anterior horn cells, causing paralysis of the atrophic type of the muscles supplied by the affected segments, and to a lesser degree with the tracts in the white matter, such as the pyramidal and spino thalamic. The posterior columns are usually spared.

**Clinical Findings** The patient may give a history of an attack of coughing, or of lifting a heavy weight, or of diving, when he is suddenly overcome with paralysis. There may be preliminary pains or numbness and tingling in the neck or shoulders.

**On Examination** (the cervical type) The patient is conscious. There is paralysis of the arms and legs, of a flaccid type at the onset, with loss of deep reflexes, constipation and retention of urine. In some cases the paralysis is very slight, but there is dissociated anæsthesia with loss of temperature and pain sensations in the hands and arms. A band of hyperæsthesia may be detected at times at the site of the level of the lesion. There is no disturbance of joint, muscle, vibration or touch sensations. In a severe case there is paralysis of the abdominal and intercostal muscles and respiration is carried on by the diaphragm. The cervical sympathetic may also be paralysed as in syringomyelia (see p. 415). The temperature is usually normal or subnormal at the onset. The cerebro spinal fluid. This is normal at the onset, but later may be yellow (xanthochromia) from blood pigments.

**Differential Diagnosis** *Hæmatomyelia* must be differentiated from meningeal hæmorrhage, acute transverse myelitis, and from syringomyelia. The onset is more gradual in syringomyelia and also in myelitis, and in the latter the temperature is raised. Meningeal hæmorrhage is accompanied by severe pains in the back and arms or legs, and often by twitching of the muscles.

**Course and Complications** If the hæmorrhage spreads, death may occur rapidly from respiratory failure. In other cases, after about a week the lower limbs may show a spastic paraplegia with loss of sphincter control, exaggerated reflexes and an extensor plantar response, while an atrophic paralysis develops in the hands and arms. The Brown Séquard syndrome may also be found, with loss of pain and temperature sensations on the side of the body opposite to the lesion and paralysis of the leg and trunk on the same side as the lesion in the cord. If the bleeding is arrested a gradual improvement occurs, with some residual lesions, resulting in weakness of the hands or legs and dissociated anæsthesia at various parts of the body. Complications include cystitis and bed sores.

**Prognosis** This is, on the whole, favourable as regards life, but some permanent disability is likely to persist.

**Treatment** The patient must be kept absolutely at rest in bed, and he may with advantage lie either prone or on his side. An ice bag should be applied over the spine at the site of the lesion. A subcutaneous injection of morphin sulph. gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  tends to calm the patient and slow the circulation, and is therefore of value. Subsequently the affected muscles should be massaged.

### Myelitis (*Myelomalacia*)

**Definition.** Inflammation and degeneration of the spinal cord.

**Etiology.** Acute myelitis may be met with under a variety of conditions. Thus it may result from exposure to cold, from trauma without visible external injury as after diving with the neck flexed, or from exposure to the concussion of shells. It may also be a manifestation of acute anterior poliomyelitis. In some cases it occurs as a complication of illnesses such as influenza, enteric fever, dysentery, measles, syphilis or gonorrhœa. Compression myelitis is due to pressure outside the cord and is considered separately (see p. 403). Acute suppurative myelitis is a rare complication of bronchiectasis and infective endocarditis, of tuberculous or suppurative spinal caries, of purulent meningitis, and of an infected meningocele or dermoid cyst. Chronic myelitis is usually due to syphilis.

**Pathology.** In some cases toxins or micro-organisms are carried to the cord by blood-vessels or lymphatics. There is, in all cases, interference with the blood supply to the cord, usually due to thrombosis, so that degeneration ensues. In acute myelitis the cord is swollen at the affected site, which may be located to a transverse area of the cord, usually in the mid-thoracic region; more rarely there is a diffuse myelitis extending up and down the cord, or disseminated patches are found. On section, the cord is soft, and no distinction can be made between the grey and white matter. On microscopical examination the nerve cells are found to have undergone chromatolysis and the nerve fibres are degenerated. In chronic myelitis the cord is smaller than normal and pale.

**Clinical Findings.** *Acute Myelitis.* The onset is usually comparatively sudden. There may be a history of any of the antecedent conditions described above. The patient is usually a young adult, and he may notice a preliminary sense of numbness or tingling in the feet or legs, or of pains in the legs or body, or of a sense of girdle constriction round the body at the level of the lesion.

**On Examination: Thoracic Transverse Myelitis.** The legs are found to be paralysed; at the onset the paralysis is of the flaccid type with loss of the knee and ankle-jerks. The abdominal reflexes are lost, but the epigastric reflex is preserved if the lesion is below the 9th thoracic segment. Priapism may be present. There is retention of urine and of feces. Sensation is lost over the legs and lower part of the body, but at the level of the lesion a zone of hyperæsthesia may be found. Later the paralysis becomes spastic, the extensor muscles being affected more than the flexors, the deep reflexes of the legs are exaggerated and the plantar response becomes extensor.

**Low Cervical Transverse Myelitis.** This is more rare. The arms and legs are paralysed, and later, while the legs become spastic, the paralysis of the arms is of the flaccid atrophic type. The cervical sympathetic may be involved on one or both sides, with enophthalmos

and constriction of the pupil. Sensation is lost below the level of the lesion.

*Lumbar Myelitis* There is flaccid paralysis of the legs, with loss of the knee and ankle jerks and of the plantar response. The latter may at times be extensor. The bladder and rectum become incontinent. The temperature is raised in cases due to infection.

The cerebro spinal fluid. In any type this may show an increase of globulin and of cells.

*Acute Diffuse Myelitis* There is an ascending paralysis which spreads up the legs to the trunk and arms, together with an ascending anæsthesia. There is loss of sphincter control. Bed sores and cystitis are very liable to develop.

*Chronic Myelitis* The paralysis is of gradual onset, with first weakness and stiffness in the legs, and later sensory loss, exaggeration of the deep reflexes and an extensor plantar response (see Spinal Syphilis p 387).

*Differential Diagnosis* In spinal thrombosis or hæmorrhage the onset of the paralysis is generally more rapid than in myelitis, and there is no fever. In acute infective polyneuritis there is a flaccid paralysis, pains in the legs are usually present, there is no loss of sphincter control, and the sensory disturbance is less marked than in myelitis. Landry's paralysis (see p 410) is by some considered to be indistinguishable from acute infective polyneuritis. In hysterical paraplegia other stigmata of hysteria can usually be detected.

*Course and Complications* The course in acute myelitis is very variable, in the diffuse variety the lesion may rapidly spread up the cord and death ensue from paralysis of the muscles of respiration. In transverse myelitis there is a tendency to recovery, which is, however, rarely complete. Some degree of spastic weakness of the legs is liable to persist, with contractures of the flexor muscles, so that the patient has to walk with the aid of two sticks. Impotence is liable to occur as a sequela. Complications include cystitis, pyelitis, pyelonephritis, and bed sores.

*Prognosis* In the diffuse and high cervical type death usually occurs in a day or so, in other cases the prognosis is as described above.

*Treatment* The patient should be nursed on a water bed and great attention paid to the skin to prevent the formation of bed sores. The skin must be kept very clean, thoroughly dried, powdered with starch and zinc oxide, pressure points must be rubbed with methylated spirits, and the heels allowed to rest in rings. All crumbs must be removed from the bed. The posture should also frequently be changed. The bowels should be emptied every other day with an enema, if necessary, and a watch kept on the bladder. If there is retention of urine it may be possible to empty the bladder by suprapubic pressure, otherwise catheterisation every 8 hours is required. Alternatively, an apparatus can be used to produce automatic tidal drainage of the bladder. A 1.5% solution of boric acid is run in from a container through a drip-bulb into the bladder. An automatic siphon empties

the bladder when a certain pressure is reached and the process repeats itself. The catheter need only be changed once a week (see Laurie and Nathan, *Lancet*, 1939, *ii*, 1072). The urine should be kept acid by the administration of acid sod. phosphate gr. 30 to 60 twice a day. If the Wassermann reaction is positive a course of iodides and mercury should be given (see p. 388).

### Compression Myelitis

**Etiology.** In the majority of cases the compression of the cord is produced gradually. It may be due to affections of the vertebræ, such as tuberculous or syphilitic caries, Paget's disease, vertebral tumours, such as primary or secondary sarcoma and secondary carcinoma, an exostosis, osteoma or chondroma. It may also occur as a complication of rheumatoid spondylitis (see p. 614). Tumours of the meninges or nerve roots may compress the cord, such as an extradural sarcoma or an intradural sarcoma or fibroma. *Leukæmia* and *Hodgkin's* disease are comparatively rare causes. Syphilitic pachymeningitis hypertrophica may compress the cord, usually in the cervical region. A hydatid cyst or a simple meningeal cyst will cause pressure. It is rare for an aortic aneurysm to erode the spine sufficiently deeply to compress the cord. With a fracture dislocation of the spine the compression is rapidly produced. This is a surgical condition.

**Clinical Findings.** *Affections of the Vertebræ:* The patient is often a child if the lesion is one of tuberculous caries, and in other cases he is generally an adult. Pain is complained of in the back, and often very severe root pains are present, radiating in a segmental distribution round the body or along the limbs, due to posterior nerve root involvement. Slight movement of the spine or coughing may cause very severe agony. Later, weakness with spasticity occurs in the legs, and numbness or tingling.

**On Examination:** In caries angular curvature of the spine is often seen, and local tenderness on tapping may be found over one or more vertebræ. In the early stages a zone of hyperæsthesia may be present, corresponding with a segmental distribution of the cord. Later there may be anæsthesia over an area in which severe pain is felt (*anæsthesia dolorosa*). Motor signs sometimes occur before the sensory ones, and when present resemble those described above for transverse myelitis, there being spastic weakness of the legs, exaggerated deep reflexes and an extensor plantar response. With progressive pressure the extensor muscles are first affected, the legs being spastic and extended, and from time to time involuntary clonic contractions occur (paraplegia in extension). Stimulation of the sole of the foot may cause not only extension of the big toe (dorsi-flexion), but also flexion of the knee and hip. Later the extensors lose their tone and the flexors are contracted (paraplegia in flexion), the hips and knees being flexed and the knee and ankle-jerks being lost. With malignant tumours of the vertebræ the excruciating root pains are a characteristic feature; there is localised vertebral tenderness but little liability to motor weakness, as death usually occurs before there is time for this to develop.

*Tumours of the Meninges and Spinal Cord:* These are usually intradural. They may be extramedullary, arising in the meninges or nerve roots, or intramedullary from the cord. The former include a sarcoma, endothelioma, myxoma, lipoma, fibroma, neuroma, gumma, psammoma, or hydatid cyst, and the latter a glioma, tuberculoma, sarcoma and rarely a gumma. The patient usually first complains of pain in one side of the back or along a limb in an area corresponding with one posterior nerve root, later, weakness appears in the leg or arm on the side of the lesion. The pain is often worse on coughing, sneezing, or at night. With an intramedullary tumour paralysis and *anæsthesia* are usually present before the occurrence of pain.

*On Examination:* There may be spastic weakness with exaggerated reflexes and loss of postural sensation in the limb on the same side as the lesion, and sensory changes with loss of pain, temperature and touch on the other side of the body. The sensory changes extend up to the level of the lesion. The cutaneous reflexes are abolished below the lesion and are normal above it. In a later stage both legs may be affected with spastic weakness, and the sensory loss is also bilateral; there is also loss of sphincter control. The cerebro-spinal fluid: In compression myelitis this shows the characteristic changes known as the *loculation syndrome*, there being an increase of protein up to 3 or 4% (normal 0.02%) below the obstruction, and a yellowish tinge is seen on looking down through a column of the fluid in a tube. This is due to extravasated blood pigment, and is called *xanthochromia*. Cells are absent or not in excess. The fluid also often clots spontaneously. In cases of spinal cord tumours it is necessary to localise the site of the lesion accurately, so that the surgeon shall know at what level to operate. This is done by a consideration of the sensory, motor and reflex changes. The lesion is usually situated one or two segments above the level indicated by the sensory loss (see Fig. 23 for segmental areas of posterior nerve roots), as the sensory fibres run up in the cord obliquely for a short distance before crossing. Further, as the fibres conveying pain and temperature sensation decussate before those of touch, with a lesion in the cord at a definite site, the cutaneous area showing the loss of touch will be lower than that showing loss of pain and temperature. The higher up the cord, the longer distance the sensory fibres travel before they decussate, so that in the upper cervical region the lesion may be situated four or five segments above the segmental level of loss of sensation in the body. Further, the spines of the vertebræ do not lie exactly over the corresponding spinal cord segments. This must be borne in mind in marking out the level over the spine for exposing the cord at operation. In the cervical region the spinal cord segment lies at a level of one spine higher, in the thoracic region the spinal cord segments 1 to 9 lie two spines higher, and from the Th. 10 downwards the spinal cord segments are three spines higher. The level of the cord lesion can also be more roughly determined by the presence or absence of the superficial and deep reflexes, which correspond with certain spinal levels, as enumerated on p. 280. The superficial reflexes are abolished below the level of the

lesion, whereas the deep reflexes are exaggerated. The various muscles also correspond with certain spinal levels, as shown in the table on p. 286, and so paralysis of definite muscles indicates the site of the lesion. X-ray examination after Lipiodol or Neo-Hydriol (ol. iodisat. B.P. Add.) injection into the cisterna magna will show the Lipiodol held up at the level of obstruction in a spinal cord compression, due either to intra- or extra-dural causes.

**Differential Diagnosis.** In diagnosing the cause of spinal compression the spine should first be examined for deformity, and also a radiogram taken. If the disease is due to an affection of the vertebræ it can usually be determined in this way. The presence of a primary malignant growth elsewhere in the body and of severe root pains is very suggestive of a malignant deposit in the vertebræ. In meningeal tumours the diagnostic features are the root pains and subsequent spastic paralysis, at first unilateral and later bilateral, with sensory disturbances. It is not usually possible to differentiate clinically between a meningeal and spinal tumour. When pain is the most prominent symptom a diagnosis of rheumatism or neuritis is liable to be made, unless a careful examination is carried out to exclude tumours or compression myelitis.

**Course and Complications.** The course depends upon the cause of the compression. Thus in malignant growths of the vertebræ the course is rapidly progressive, whereas a meningeal tumour may exist for several years. In spinal caries the course is very variable and often the disease is arrested.

**Prognosis.** This must vary with the cause, as described above.

**Treatment.** Spinal caries is a surgical disease, and is treated by rest and exposure to sunlight. In secondary malignant vertebral tumours nothing can be done to cure, but pain may be relieved by division of the posterior nerve roots. Many spinal and meningeal tumours can be removed by operation. The Wassermann reaction should always be carried out, and, if positive, a course of anti-syphilitic treatment given before an operation is performed. If there is no improvement, operation should not be delayed for more than 3 to 4 weeks, as a tumour may be present which is not a *gumma*.

### *Compression of the Cauda Equina*

**Etiology.** This may be due to a fracture dislocation in the region of the lower lumbar vertebræ, to spina bifida, or to a tumour. Extradural tumours include a sarcoma and chondroma, and intradural tumours a meningeal or perineural fibroblastoma. The cauda equina embraces the nerve roots below those arising from the second lumbar segment of the cord.

**Clinical Findings.** The patient complains of pain in the lower part of the back or the legs, having a root distribution. There is also weakness of one or both legs, and often retention or incontinence of urine and faeces.

**On Examination:** The leg or legs show muscular weakness or paralysis, with loss of tone and wasting. The knee-jerk and ankle-jerk

are absent The plantar response is absent or flexor. Loss of sensation is found in the legs and perineum

**Prognosis** This varies with the cause, when due to trauma recovery often occurs

**Treatment** If a tumour is suspected an exploratory laminectomy should be performed Otherwise the treatment is as for compression myelitis

### Acute Poliomyelitis and Polioencephalitis (Heine Medin Disease Infantile Paralysis)

**Definition** An acute disease characterised by lesions having a special affinity for the grey matter of the anterior horns of the spinal cord

**Etiology** The disease is caused by a neuronotropic filtrable virus Infection takes place either by droplets disseminated by carriers or abortive cases, or by milk food or water contaminated possibly by flies Epidemic and sporadic infection occurs *Predisposing causes*

1 Age Chiefly between 2 and 5 years adolescents and young adults are sometimes affected 2 Sex Males predominate slightly 3 Season Summer and autumn but in all districts sporadic cases are constantly present 4 Locality America, Canada Australia, Scandinavia and to a lesser degree Great Britain

**Pathology** Two views are held regarding the site of infection and the mode of spread. 1 Droplet infection occurs in the naso-pharynx, the virus spreads to the brain along the olfactory nerves, then to the mid brain, spino thalamic tracts, dorsal root ganglia and anterior horn cells Experimental evidence shows conclusively that infection can occur by the olfactory route in the rhesus monkey 2 Droplet infection or food infection takes place by the mouth, the virus multiplies chiefly in the wall of the pharynx and small intestine It passes to the central nervous system through the pharyngo tonsillar or intestinal mucous membrane, travelling along autonomic nerves to the medulla or spinal cord The infection may pass along parasympathetic fibres in the VII, IX or X cranial nerves, along sympathetic fibres to the thoracic lumbar region and along parasympathetic fibres to the sacral region In neither view is it believed that infection is spread by the blood stream Evidence is accumulating to support the second view Indirect evidence in its favour includes the seasonal incidence, when droplet infection is likely to be at its lowest. Further, in some epidemics the case incidence resembles that of a milk borne epidemic. Also the virus is excreted in the faeces It can be detected in the faeces of patients suffering from the paralytic and the common abortive types of the disease It may be present in the faeces during convalescence for over 3 months, and it may also be detected in sewage The commonly occurring lumbar type of infection is more easily explained if the virus gains access to the nervous system from the alimentary tract rather than from the nose, and the bulbar type is due to infection in the tonsillo-pharyngeal region and spread along the pharyngeal nerves Experimental evidence shows that chimpanzees, whose olfactory



eruption may be seen on the affected limb. The bowels are usually constipated. If the lesion is situated in the mid thoracic region there may be a definite transverse myelitis, with sphincter paralysis, flaccid paralysis below the level of the lesion and loss of deep reflexes. The flaccidity is later followed by a spastic paraplegia.

**Varieties** In addition to the spinal type described above, certain other varieties of the disease are recognised by Wickman. They include Abortive, neuritic, spreading (ascending or descending), meningeal, cerebellar, cerebral, brain stem and mixed types. These types will be briefly described.

- 1 *The abortive type* The disease does not progress beyond the stage of invasion of the subarachnoid space, with consequent meningeal symptoms. Recovery occurs in 2 or 3 days.
- 2 *The neuritic type* There are severe pains in the arms or legs. The deep reflexes are diminished. There is no paralysis.
- 3 *The spreading type* This resembles Landry's paralysis (see p. 410). The paralysis may spread up or down the cord in the space of a few hours, and if the respiratory muscles are all paralysed death results.
- 4 *The meningeal type* This resembles the abortive type, but the symptoms are more marked and more prolonged. There is no paralysis.
- 5 *The cerebellar type* The patient has intense headache, vertigo, vomiting on moving the head, ataxia of speech and of spinal muscles, and nystagmus.
- 6 *The cerebral type* The onset is with generalised convulsions persisting for several hours, followed by hemiplegia or paraplegia, with spasticity and an extensor plantar response. There may also be mental deterioration and athetotic or choreic movements.
- 7 *The brain stem type* The VII, VI and III nerves are most commonly affected. In some cases the lesions are bulbar, there being dysphagia, dysarthria, dysphonia, disturbances of breathing and irregularity of the heart. Bulbar poliomyelitis has been frequently reported following tonsillectomy performed during epidemics of poliomyelitis.
- 8 *Mixed types* A combination of cerebral and spinal lesions may be present. Thus one leg may show a spastic and the other a flaccid paralysis.

**Differential Diagnosis** Sporadic cases may be difficult to diagnose at the onset. There are no distinguishing features during the prodromal period. In the meningeal stage the cerebro spinal fluid usually shows the characteristic findings described above. In tuberculous meningitis the chloride content is reduced, in bacterial meningitis the sugar content is lowered and the causative organism is present, and in encephalitis lethargica polymorphonuclear cells are not usually found. In encephalitis lethargica paralyses are not so common and usually not so acute in onset, and meningeal symptoms are rare. In polioencephalitis the onset is usually more acute, more than one member of a family is likely to be affected, the patient is not usually over the age of 30, the temperature is generally higher, and the course of the acute stage of the disease is shorter than in encephalitis lethargica. When paralysis has appeared and the muscles are tender and painful, the disease may be mistaken in infants for scurvy, acute rheumatism, osteomyelitis or syphilitic epiphysitis. The neuritic form is differentiated from multiple neuritis

by the fever and by special groups of muscles being affected. The spreading type closely resembles Landry's paralysis (see p. 410).

**Course and Complications.** Although in the majority of cases the paralysis is maximal at the outset, the paralysis may spread in a few hours from muscle group to muscle group. Relapses are not unknown during the course of the illness, with rise of temperature and further paralysis of muscles, but they are not usually serious. Some of the affected muscles recover completely, others undergo partial recuperation, and others may be permanently paralysed. Scoliosis may result from a minor attack of poliomyelitis which has picked out certain spinal muscles. The bones of the affected limbs do not usually grow as well as normal, with subsequent shortening of the leg or arm. Contractions and deformities result from permanently damaged muscles. Progressive muscular atrophy has occasionally been noted as a late sequela.

**Prognosis.** The mortality rate in an epidemic varies between about 10 and 20%. Complete recovery may be expected even if all four limbs are paralysed, if the superficial and deep reflexes persist. Muscles which are painful and tender are more likely to recover than those which are insensitive. The mortality is very high when the medulla is affected, and spreading infection is usually very serious, death resulting from respiratory paralysis or from bronchopneumonia. A muscle which responds to faradisation usually recovers, but the prognosis is serious if the reaction of degeneration is present. Some degree of recovery may be expected for a year after the onset of the disease.

**Treatment. Prophylactic.** On the supposition that the infection is carried in the naso-pharynx contacts should gargle twice a day with 1 in 5,000 solution of potassium permanganate or with normal saline. They may also be given an intramuscular injection of 5 mls of convalescent serum obtained from a patient within 10 days of his becoming afebrile. This should afford protection for about 3 weeks. Attempts at active immunisation using an inactivated vaccine have not proved successful, and injections of an attenuated virus are dangerous. If an outbreak occurs in a boarding school there are arguments for and against sending the pupils home. The risk of so doing consists in planting a carrier in a new zone, where he will spread the infection. The child must be kept in quarantine for 14 days. On the other hand, if healthy children are kept in the school they run the risk of contracting the disease from a carrier or from infected food or water.

**Curative.** The patient should be isolated for three weeks after the temperature has become normal. It is very doubtful, however, if a patient is infective when the nervous paralytic signs have appeared, and case to case infection in a hospital is practically unknown. As the virus is eliminated in the faeces strict precautions should be taken with regard to their disinfection and disposal. The patient should be kept warm in bed, and if the serum from a convalescent patient, or from a person who has previously had the disease, is available, it should be used during the pre-paralytic period. Lumbar puncture is then performed on the patient, 10 to 15 mls of spinal fluid are removed, and a slightly smaller volume of the serum is injected intrathecally.

Thirty mls may also be injected intravenously. Lumbar puncture during the meningitic stage usually relieves temperature, headache and muscular pains, and may be repeated for the first 2 or 3 days. Encouraging results have been reported in severe cases by the combined use of Sulphapyridine (M & B 693) and convalescent serum. In a severe case in an adult 2 to 3 G of M & B 693 soluble diluted to 20 or 30 mls with normal saline are injected intravenously and 20 mls of convalescent serum are given intramuscularly. The Sulphapyridine is repeated every 4 hours for 4 doses, and a second injection of serum is given 10 hours after the first. The treatment is repeated if necessary on the second and third days. In mild cases the Sulphapyridine is given by mouth, first 2 G and then 1 G every 4 hours until the temperature has fallen to normal. Great care must be taken to prevent stretching of paralysed muscles. The limbs must be placed in such a position that the affected muscles are relaxed and the position maintained by pillows, sandbags, or celluloid splints. Hot wet flannels may be applied to the affected muscles. When the respiratory muscles are affected, artificial respiration may be carried out for prolonged periods automatically by the use of the Drinker respirator. Pain may be relieved by the administration of aspirin in doses of gr 5 to 10 t d s, according to the age of the patient. The muscles should not be massaged during the period of active infection, and no electrical treatment should be given. After 3 or 4 weeks, gentle massage may be applied. Orthopaedic treatment may improve contractures or deformities, but will not be required for at least a year after the onset of the illness.

### Landry's Paralysis

#### (*Acute Ascending Paralysis*)

**Definition** A disease characterised by acute ascending paralysis, with little or no sensory or sphincter disturbance.

**Etiology.** The cause is not known. The disease is probably due to a toxin. By some authorities Landry's paralysis is regarded as a manifestation of acute toxic polyneuritis, and by others as an acute polioencephalomyelitis. Probably both types exist. One case of acute ascending myelitis has been shown to be due to a neurotropic filtrable virus. *Predisposing causes* 1 Age 20 to 45 years 2 Sex Males predominate.

**Pathology** In some cases no changes are found post mortem, in others there is hyperæmia of the vessels of the cord, chromatolysis of the anterior horn cells, or interstitial changes in the peripheral nerves.

**Clinical Findings** The patient is usually suddenly seized with weakness, first in one leg and then in the other. There may be prodromal symptoms, such as malaise, numbness or tingling in the feet, or pains in the back or legs. The paralysis rapidly spreads up the legs. In the case originally described by Landry the shoulders, the arms and hands were next paralysed, and finally præsthesia spread to the trunk and there was dysphagia. This corresponds with a neuritic origin of the disease. In other cases, subsequently classified as Landry's paralysis, the trunk is affected after the legs, then the arms, neck, head

and finally the tongue and muscles of deglutition. Involvement of the diaphragm and intercostal muscles usually results in death from respiratory failure.

**On Examination :** Spinal motor system : There is flaccid paralysis of the legs, trunk and arms, and finally of the neck, face and head. The diaphragm and intercostal muscles may or may not be affected. The muscles do not waste, or only very slightly. Spinal sensory system : Often there are no changes ; some loss of touch sensation may be detected in the toes. The reflexes : The deep and superficial reflexes are lost. The sphincters are usually unaffected, but there is often retention of urine and of fæces from lack of muscular power. In some cases the XII, XI, VII, or III, IV and VI nerves may be affected. Trophic changes : These do not usually occur. Electrical reactions : There is no reaction of degeneration. Response to galvanism is usually lost, but faradic response remains. Lumbar puncture : The fluid is usually under increased tension. It is clear, and may contain an excess of protein and of cells. Cerebration : The mind usually remains clear until the end. The temperature is not usually raised. The spleen may be palpable.

**Differential Diagnosis.** Acute toxic polyneuritis : By some authorities this is held to be indistinguishable from Landry's paralysis. However, in the former there is more likely to be fever, pain in the limbs, with anæsthesia and wasting of muscles. The abdominal reflexes may also be present. Acute ascending myelitis : Here there are more marked sensory changes, with sphincter paralysis and the development of bed sores. Acute polioencephalomyelitis : The spread of the paralysis is more irregular, there is fever and wasting of the paralysed muscles.

**Course and Complications.** The disease is usually rapidly progressive. Deglutition bronchopneumonia may occur as a complication.

**Prognosis.** Death frequently occurs in a few hours or days. This may be due to paralysis of the respiratory muscles or of the heart, or to deglutition bronchopneumonia. If the patient recovers, there is usually no wasting or paresis of the affected muscles.

**Treatment.** When the respiratory muscles are affected the patient should be put in a Drinker respirator and hypodermic injections of strychnin. hydrochlor. gr. 1/60 and atropin. sulph. gr. 1/120 given four-hourly. Nasal feeding and inhalations of oxygen may also be required. Lumbar puncture should be made as required to relieve the increased tension of the spinal fluid.

### Disseminated Sclerosis (Primary Multiple Sclerosis)

**Definition.** A disease characterised by spastic weakness of muscles, with a progressive course often interrupted in the early stages by remissions.

**Etiology.** The cause is not known. It is thought to be due to the action of toxins or to the presence of a myelinolytic ferment in the blood. Various possible causative organisms have been described,

without sufficient evidence to justify their acceptance *Predisposing causes* 1 Age Usually between 20 and 40, rarely under 10 or over 45 2 Sex Females predominate slightly There is no familial incidence but several cases may occur in a household The disease may show itself after an attack of influenza or scarlet fever, etc It is a very commonly occurring nervous disease

*Pathology* Lesions are scattered throughout the brain and spinal cord, varying in size from a pin point or less to about  $\frac{3}{4}$  inch in diameter. The recent lesions are pinkish in colour and the old ones greyish white They are present both in the grey and white matter of the central nervous system, and the optic nerve is often affected There is degeneration of the myelin sheath of the nerve fibres, the axis cylinder usually remaining intact There is also a perivascular infiltration with lymphocytes and plasma cells and some local oedema In the older lesions there is proliferation of neuroglial tissue Secondary degeneration above or below the damaged area in the cord rarely occurs

*Clinical Findings* In the majority of cases the disease has an insidious onset, although the first symptom noted occurs suddenly The patient is a young adult who may notice weakness in one leg on walking, the foot tending to catch in the ground, or there may be numbness or tingling in a leg or vague aching Early urinary symptoms may occur, such as difficulty in beginning micturition or retention In other cases the patient suddenly loses the vision in one eye, recovery taking place in a week or so, or there may be a transient attack of double vision An attack of acute unilateral retrobulbar neuritis, lasting a week or so, may precede the onset of other symptoms by several years *The acute variety* Rarely the disease begins with an attack of hemiplegia during which the patient does not lose consciousness, or with epileptiform convulsions It is unusual for the weakness to affect the arms before the legs

*On Examination* In the early stages no abnormal signs may be found, and in such a case the patient should be re examined from time to time so that a diagnosis can be established as soon as possible A tendency to laugh easily, unnatural cheerfulness or euphoria may be noted in the early and advanced stages of the disease The signs which occur in disseminated sclerosis are as follows Weakness may be detected in certain groups of muscles such as the flexors of the ankles or of the hip There is a tendency to spasticity of the leg muscles The knee-jerks and ankle jerks are brisk, and ankle clonus and patella clonus may be present The plantar response is extensor The abdominal and cremasteric reflexes are lost. Areas of loss of cutaneous sensation may be found on the legs, the bone vibration sense is diminished or lost in the legs, joint sensation is usually lost. Pallor may be detected in the temporal halves of the optic discs In cases of loss of vision in one eye there is a retrobulbar neuritis The affected pupil is usually dilated, and does not respond well to direct stimulation by light, although it contracts consensually when a light is shone into the other eye A small central scotoma for colours may also be present Weakness of accommodation may be a troublesome

feature. A history of transitory diplopia or urinary disturbance may be obtained on questioning. In more advanced cases the triad of symptoms described by Charcot may be found. These are nystagmus, intention tremors and scanning speech. The nystagmus is a fine horizontal one. "Jelly nystagmus" is almost a diagnostic sign when present. On ophthalmoscopic examination the fundus appears to quiver rapidly. The intention tremors are seen when the patient is asked to lift up a glass of water or perform some other movement, and become more marked towards the completion of the act. The speech is slow and the syllables are pronounced somewhat abruptly. There may be difficulty in beginning micturition, or incontinence. In the later stages contractures occur in the spastic muscles, and there are marked tremors of the head, neck and arms, so that the patient is bed-ridden and unable to look after herself. Other types include a cerebellar variety characterised by vertigo, ataxia and nystagmus, and a cerebral one, with progressive hemiplegia. The blood: The Wassermann reaction is negative. The cerebro-spinal fluid: This may be normal. In about 30 to 70% of cases a paretic gold curve is obtained (see Fig. 25). There may also be an excess of cells and globulin.

**Differential Diagnosis.** In the early stages the disease is very liable to be mistaken for hysteria, or the retrobulbar neuritis may be thought to be due to sphenoidal sinusitis. When there is spastic paraplegia, spinal syphilis must be excluded, and local pressure due to bony changes or a cord tumour. The ataxy must be distinguished from that due to Friedreich's ataxia, in which the deep reflexes are depressed.

**Course and Complications.** In the early stages remissions are a very characteristic feature, the weakness of the limb disappearing and reappearing later. Fresh symptoms appear at different parts of the body as new lesions develop in the central nervous system. Arrest may occur at any stage, or the patient may rapidly become bed-ridden. Complications include septic infection of the urinary tract and inter-current lung infections.

**Prognosis.** This is very unfavourable, but death may not occur for 10 years or later after the onset.

**Treatment.** There is no known cure. Any obvious septic focus should be eradicated. A course of neoarsphenamine should be given, starting with 0.1 G., and increasing to 0.6 G., six intravenous injections in all. Subsequently mercurial inunctions and iodides are given as for nervous syphilis (see p. 388).

Treatment by protein shock, using a T.A.B. vaccine (see p. 612), or by malarial therapy (see p. 393), has not proved of great value.

General measures include the avoidance of fatigue and the use of hot baths to diminish spasticity.

### Syringomyelia

#### (Syringomyelomeningocele)

**Definition.** A disease characterised by a peculiar sensory disturbance, muscular wasting and trophic lesions, due to cavity formation and neuroglial overgrowth in the cord and mid-brain.

**Etiology** The cavity formation is generally believed to be due to a developmental defect, the central canal of the cord not closing properly. It is possible that in some cases there is first an overgrowth of neuroglial tissue, which breaks down to form a cavity. **Predisposing causes**  
 1 Age Usually the symptoms are first noted between 10 and 30 years.  
 2 Sex The incidence is about equal. Trauma to the spine may be a predisposing cause in some cases.

**Pathology** The spinal cord shows characteristic changes. The dura is usually normal, but the pia may be thickened. The cavity is generally situated posterior to the central canal in the posterior commissure. It is most likely to be present in the lower cervical and upper thoracic regions, running up and down the cord for a variable distance. Thus it may extend for a few segments, or run the length of the cord and into the medulla. The cavity may branch and vary in size from a small hole to one occupying the greater part of the transverse section of the cord. The cord may be distended and cause pressure on the vertebræ. The cavity may contain a thin watery or glairy fluid and may communicate with the central canal of the cord and be lined with ependymal cells. The neuroglial overgrowth occurs around the cavity and isolated masses of neuroglial tissue may be present at varying sites in the cord. The cavity and the surrounding neuroglial overgrowth are most likely to interfere with the nerve fibres entering from the posterior root on one or both sides which convey impulses of pain and temperature, and cross the cord in the anterior commissure to ascend in the spinothalamic tract on the other side. If the cavity extends further outwards it may involve the fibres entering from the posterior root which convey impulses of touch and ascend on the same side of the cord. Extension forwards into the grey matter causes atrophy of the anterior horn cells, and lateral extension may interfere with the pyramidal tract.

**Clinical Findings** The onset is usually insidious. The patient may first notice pains in the hand or arm, or weakness of the hands, or clumsiness in fine movements. In other cases he burns his fingers with a cigarette without feeling heat or pain, or a cut on the finger is painless. If there is gross dilatation of the cervical cord, severe pain may be felt in the neck.

**On Examination** In an established case of the cervical type the following changes may be found. There is usually kyphoscoliosis in the thoracic region, and a spina bifida may be present. Spinal motor system. There is wasting of the small muscles of one or both hands, so that a claw hand or monkey hand is seen. The muscle wasting may extend to the arm and shoulder girdle, with winging of the scapula. In muscles which are actively wasting, fibrillary contraction may be seen. The deep reflexes of the arms may be increased or diminished, and a reaction of degeneration may be present in the affected muscles. There is often spastic weakness of the legs, with increased knee and ankle jerks and an extensor plantar response. Spinal sensory system. Sharply defined areas of dissociated anaesthesia are present on the hands, arms, neck, trunk, etc. There is loss of sensation to heat,

cold and pain, whereas touch sensation, the discrimination of two points, joint sensation, muscle sensation, and the vibration sensation are preserved. In some cases there is a complete hemi-anæsthesia to pain and temperature senses on the opposite side of the body below the level of the lesion, due to involvement of the spino-thalamic tract. Trophic changes include painless swelling of the shoulder, elbow or wrist, similar to the Charcot's arthropathy of tabes dorsalis. The bones of the arm may break spontaneously. The hands may be enlarged and thick, *main succulente*, owing to thickening of the subcutaneous tissues; painless ulcers or subcutaneous whitlows may occur on the fingers, patches of red or blue skin may be seen on the hands. Involvement of the cervical sympathetic on one side is indicated by Horner's syndrome, i.e., enophthalmos, narrowing of the palpebral fissure, contraction of the pupil and absence of sweating on one half of the face. Other types of the disease are more rarely met with. They include: *The thoracico-lumbar variety*: There is wasting of the muscles of the pelvic girdle and legs, and dissociated anæsthesia of the legs. *The sacro-lumbar variety*: The small muscles of the feet and the leg muscles waste. Trophic lesions may be seen on the feet. There is dissociated anæsthesia of the feet or legs. There is usually loss of control of the bladder and rectum. The deep reflexes are often exaggerated in the legs and the plantar response is extensor. *The bulbar type (syringobulbia)*: Bulbar symptoms may occur in the cervical type, or independently. The lesions are usually unilateral, such as laryngeal paralysis, dysphagia, wasting of half of the tongue, nystagmus and ocular paresis, anæsthesia of half of the face, or facial weakness. Further, certain clinical types are described, such as: 1. The classical type depicted above under the cervical variety. 2. The motor type, in which motor symptoms predominate and resemble those of amyotrophic lateral sclerosis. 3. The sensory type, which may be mistaken for hysteria. 4. The trophic type, here all sensation may be lost in the hands and painless whitlows and ulcers occur (Morvan's disease). 5. The tabetic type, in which the lesion spreads into the posterior columns, and there are loss of knee-jerks, Rombergism, severe pains and arthropathies of the legs.

**Differential Diagnosis.** The diagnosis of syringomyelia is established by the presence of dissociated anæsthesia. Difficulty occurs in cases in which motor symptoms predominate or are first noted. Then other conditions, such as a cervical rib, progressive muscular atrophy, amyotrophic lateral sclerosis, disseminated sclerosis and lateral sclerosis, may require exclusion. Horner's syndrome may also result from a cervical rib, a spinal cord tumour at the level of C.7 to Th.1, cervical pachymeningitis, an aortic aneurysm, mediastinal tumour, apical lung carcinoma, trauma, etc. In hæmatomyelia the signs may closely resemble those of syringomyelia, but in the former the onset is more sudden, and, as the blood is absorbed, the signs gradually disappear.

**Course and Complications.** Syringomyelia usually pursues a slowly progressive course lasting from 10 to 20 years or longer, but for a considerable time the symptoms and signs may remain stationary.



Hæmorrhage may take place into the cavity, causing pain or paraplegia. Other complications include cystitis, pyelonephritis and pulmonary tuberculosis

**Prognosis** This is unfavourable. Death is usually imminent if there are extensive mid brain lesions

**Treatment.** No cure is known. Care should be taken to protect anæsthetic areas from injury. Mercurial inunction is recommended, with iodides by mouth, as in the treatment of syphilis (see p 388), but the value of this treatment is very doubtful. Operations to drain the cavity or relieve pressure on the vertebræ are often fatal. Deep X ray treatment is not usually successful. Pains may be usually relieved by aspirin or phenazone, gr 5 t d s

### *Subacute Combined Degeneration of the Cord*

**Definition** A disease characterised by degeneration of the posterior and lateral columns of the cord, with usually anæmia of the pernicious type and achylia gastrica

**Etiology** The cause is not known. There may be a gastric deficiency factor, analogous to that thought to be present in pernicious anæmia (see p 187), associated with a congenital abiotrophy of nerve tissue. *Predisposing causes* 1 Age 30 to 65 years 2 Sex The incidence is equal. In some cases there is a familial occurrence of the disease

**Pathology** The lower thoracic part of the cord is usually the first site of the lesion. There is degeneration of the white matter, which begins in the posterior or lateral columns in irregular patches. These tend to fuse and to spread up and down the cord in a funicular manner, and the internal capsule of the brain may be affected. The medullary nerve sheaths and later the axons in the cord degenerate. There is very little tendency to a proliferation of neuroglial tissue. The peripheral nerves may show degeneration, but the grey matter of the cord and the nerve roots themselves are not usually affected. Changes characteristic of pernicious anæmia (see p 488) may be found in other parts of the body

**Clinical Findings** The patient is usually an adult of middle age, who complains of the gradual onset of peculiar feelings in the legs or arms, such as numbness, pins and needles, burning or pains. These sensations are frequently first felt in the toes or fingers, and later extend up the limbs. Sharp "lightning" pains may also be felt in the limbs, or a "girdle" sensation around the trunk. The legs gradually feel heavy and walking is an effort. When the eyes are closed, or when the patient is in the dark, he may feel definitely unsteady. Impotence is the first symptom in some cases. Often the blood shows evidence of pernicious anæmia when the nervous symptoms appear, although it is rare for subacute combined degeneration to develop in a patient who has been adequately treated for pernicious anæmia. Many cases have, however, been recorded in which no definite anæmia was detectable when the nervous symptoms appeared. An acute onset is more rare, with fever, vomiting and diarrhoea, and pains in the back and legs

*On Examination:* Spinal sensory system: Changes are usually first detected here. Anæsthesia for pain, temperature and touch may be found in the periphery of the legs or arms in a stocking or glove area. Vibration sense disappears in the tibiæ. The joint sense of the big toe is lost. The calf muscles may be unduly tender on pressure. On the trunk, segmental areas of anæsthesia may be detected. Spinal motor system: The muscles of the legs become weak and their tone increased, but in some cases there is a flaccid paresis. There may be wasting of the small muscles of the hands. The reflexes: The knee and ankle-jerks are usually exaggerated and an extensor plantar response is obtained. In the flaccid type of paresis the knee and ankle-jerks are sluggish, but the plantar response here is usually extensor. The abdominal reflexes are generally exaggerated. The sphincters become paralysed late in the disease. Co-ordination: Loss of postural sense gives rise to inco-ordination and Rombergism. The gait: This is ataxic. Trophic changes: Oedema of the subcutaneous tissues and bed sores are liable to develop in advanced cases. The cranial nerves: In some instances there is primary optic atrophy or optic neuritis. Nystagmus may also be present. Involvement of the cervical sympathetic will give rise to enophthalmos and constriction of the pupil, usually on one side. Mentality: There may be a gradual lowering of the mental calibre of the patient. The cerebro-spinal fluid: This is normal. Electrical reactions: There is usually no reaction of degeneration, but a gradual diminution of response to electrical stimuli occurs. *General examination:* The tongue is often smooth and shiny. The blood: Changes typical of pernicious anæmia (see p. 488) are generally found in cases of some duration. The fractional test meal shows achylia gastrica, as in pernicious anæmia. The spleen may be palpable. During the course of the disease an irregular degree of fever may be noted.

*Differential Diagnosis.* In the early stages the symptoms and signs may be those of peripheral neuritis, and the weakness of the legs and stocking or glove anæsthesia may lead to a diagnosis of hysteria. The occurrence of an extensor plantar response, however, puts this diagnosis out of court. When the posterior columns are chiefly affected, the case resembles one of tabes dorsalis, but this again is excluded by an extensor plantar response, the achylia, and the normal cerebro-spinal fluid. Involvement of the lateral columns of the cord causes the disease to resemble disseminated sclerosis, Friedreich's ataxia, amyotrophic lateral sclerosis, primary lateral sclerosis, or a cord tumour. The combination of evidence of a posterior and lateral column cord lesion, with achylia gastrica and a blood count resembling that of pernicious anæmia, enables the diagnosis of subacute combined degeneration to be made.

*Course and Complications.* The disease usually pursues a gradually progressive course. In some cases it may be divided into three stages: first that of ataxic paraplegia; this is followed by spastic paraplegia with marked evidence of anæsthesia, and finally there is a flaccid paraplegia with loss of sphincter control. It is comparatively rare

for the disease to pursue a rapid course of a few weeks or months. Complications include cystitis, pyelitis, pyelonephritis and bed sores.

**Prognosis** Death usually occurs in 2 to 3 years from the onset of symptoms. The outlook appears, however, to have been improved by the liver treatment and by iron therapy, as cases have now been recorded in which the patient has been enabled to return to work, and such signs of organic nervous lesions as the extensor plantar response and sensory loss have disappeared. The blood count also returns to normal.

**Treatment** The patient should be given 5 mls of a liver extract, such as Hepatex or Hepastab, intramuscularly, daily until he can walk, and subsequently an adequate maintenance dose must be injected every 1, 2, 3 or 4 weeks (see p. 490). The administration of Bland's pill (pil. ferri carbonatis B.P.) gr. 50 t.i.d., for several months has also produced extremely good results in a series of cases showing no anaemia.

### Friedreich's Ataxia

#### (Hereditary Spinal Ataxia)

**Definition** A disease characterised by ataxia, due to sclerosis of the posterior, spino cerebellar and pyramidal tracts of the cord. There is a familial or hereditary tendency.

**Etiology** The cause is unknown but it may be due to a premature degeneration (biotrophy) of certain nerve fibres. *Predisposing causes*

1 **Heredity** The disease tends to occur in several members of a family, and at times in several generations of the same family. 2 **Age** Usually between 5 and 15 years, rarely it does not develop until about the age of 30. 3 **Sex** Both sexes are equally affected.

**Pathology** The spinal cord may appear smaller than normal. There is atrophy of the nerve fibres of the following tracts. The posterior columns, the spino cerebellar tracts of Gowers and Flechsig, and the direct and crossed pyramidal tracts. Clarke's column is also affected. Sclerosis results from a secondary overgrowth of neuroglial tissue. The changes are most marked in the lumbo-sacral part of the cord. The cerebellum is usually normal although some cellular degeneration may occur. In some cases congenital pulmonary stenosis is also present.

**Clinical Findings** The onset is insidious. The mother may notice a gradually developing deformity of her child's feet, or that there is difficulty and clumsiness in walking. The latter may be intensified in the dark.

**On Examination** The nervous disease case sheet described on p. 283 can be filled in as follows —

### Case Sheet

#### (Friedreich's Ataxia)

**Age** 5 to 15 **Sex** Male or female

**Cerebration** The child may be backward **Speech** Slurred

**Cranial Nerves** I Normal II Optic atrophy may occur III, IV and VI Lateral nystagmus may be present V (a) Motor Tremors of masticatory muscles may occur (b) Sensory Normal VII (a) Motor Twitching of the facial muscles may occur (b) Sensory

Normal. VIII Normal. IX, X and XI Usually normal. XII Tremors of tongue may occur.

*Spinal Motor Nerves.* (a) Power. There is weakness of the legs and, perhaps, of the arms. (b) Wasting. This may occur late in the weak muscles. (c) Tone. The affected muscles are usually flaccid. (d) Contractions. Contractures of the foot muscles result in pes cavus, hyperextension of the big toe and hammer toes. Kyphoscoliosis is also due to muscular action. Tremors of the head, neck and trunk are often seen and intention tremors of the hands and arms, so that there is difficulty in picking up fine objects.

*Spinal Sensory Nerves.* (a) Cutaneous sensation: This is usually normal. It may be slightly blunted on the feet. (b) Joint sense: This is often normal, but may be lost later in the big toe. (c) Kinæsthetic sense: Often lost later. (d) Muscle and tendon sense: This may be normal or lost in the legs. (e) Nerve sensation: This is usually normal. (f) Stereognostic sense: Usually normal. (g) Vibration sense: This may be lost over the tibiæ. (h) Subjective: There are usually no pains.

*The Reflexes.* (a) Superficial. 1. Conjunctival and corneal: Normal. 2. Palatal: Normal. 3. Pharyngeal: Normal. 4, 5 and 6. Epigastric, abdominal and cremasteric are lost late in the disease. 7. Plantar: Extensor. (b) Deep. 1. Pupil: Reaction sluggish to light and accommodation. 2. Jaw-jerk: Not present. 3, 4 and 5. Biceps, triceps and supinator jerks may be absent. 6 and 7. Knee-jerk and ankle-jerk: Lost. (c) Visceral. Normal.

*Co-ordination.* There is often inco-ordination of the arms and legs, but Rombergism may not be present.

*Trophic Changes.* Bed sores may occur in the terminal stages. *Gait: Reeling.* The patient stands on a wide base. Static ataxia is often seen, the patient swaying when he is standing still. *Electrical reactions: Normal.* *Lumbar puncture: Fluid normal.*

*Differential Diagnosis.* Juvenile tabes is excluded by the familial incidence, foot deformity, extensor plantar response and absence of pupillary changes in Friedreich's disease. In disseminated sclerosis the knee-jerks are exaggerated, and the age incidence is usually later. In Marie's hereditary ataxia the onset again is later, the knee-jerks are exaggerated, and there are no deformities of the feet.

*Course and Complications.* The course is usually slowly progressive, the patient eventually being confined to bed.

*Prognosis.* In some cases the disease is arrested, and frequently the patient may live for over 30 years from the onset.

*Treatment.* There is no known cure. The limbs should be massaged and walking exercises encouraged. Special boots are usually required.

### Spino-cerebellar Ataxia

#### (Marie's Hereditary Cerebellar Ataxia)

This disease resembles in many respects Friedreich's ataxia. The onset is usually later, generally after the age of 20 years. It shows both a hereditary and a familial incidence. There may be some

degeneration of cells in the cerebellum, but the chief lesion is in the spino cerebellar tracts of the cord, especially in the dorsal cerebellar tract of Flechsig. The gait is of a reeling character, there is inco-ordination of the legs and later of the arms. The speech is slurred, and optic atrophy and nystagmus may be present. The knee jerks are usually exaggerated. The disease differs from Friedreich's ataxia in several points such as the absence of deformity of the feet or of kyphoscoliosis, the presence of muscular hypertonia and the exaggerated knee jerks. Optic atrophy is more common than it is in Friedreich's ataxia. Another type of spino cerebellar ataxia is known as *Sanger Brown's ataxia*. This also resembles Friedreich's ataxia. The chief distinguishing points of Sanger Brown's ataxia are. Optic atrophy is generally present, there is no nystagmus and the plantar reflexes are flexor. It also usually develops at a later date than does Friedreich's ataxia.

### Peroneal Muscular Atrophy

(*Charcot Marie Tooth Type of Muscular Atrophy*    *Progressive Neural Muscular Atrophy*)

**Definition.** A disease characterised by wasting of the small muscles of the feet and hands, and the muscles of the distal parts of the extremities associated with lesions in the central nervous system.

**Etiology.** The cause is unknown. *Predisposing causes.* 1 There is a familial tendency and it appears to be transmitted by females. 2 Age. Between 5 and 10 years or in early adult life. 3 Sex. Males predominate slightly.

**Pathology.** Degeneration of the anterior horn cells occurs in the sacral, lower cervical and upper thoracic regions of the cord. Degeneration may also be present in the cells of Clarke's column and in the posterior and postero lateral columns of the cord. The motor nerves supplying the affected muscles atrophy, and fibrosis occurs in the wasted muscles.

**Clinical Findings.** The disease starts gradually, the patient experiencing difficulty in walking as the feet tend to drop and become inverted. A condition of bilateral talipes equino varus thus develops. Later as the muscles in the legs are affected, there is greater difficulty in walking. After a few years wasting is noticed in the intrinsic muscles of the hands with tremors, and the muscles of the forearms are subsequently affected.

**On Examination.** During the active stages of the disease, fibrillary twitchings are seen in the muscles which are wasting. Contractures tend to occur so that the feet are clubbed and the hands clawed. The thighs are not affected, except that in some cases there may be muscle wasting just above the knees, the upper arm, trunk, neck and face muscles are unaffected. The limbs assume the shape of a bottle with the neck downwards. The knee jerks are brisk, but the ankle jerks are lost. The superficial reflexes are present, but the plantar response is sluggish or difficult to obtain owing to the contractures. Sensation may be normal or there may be varying degrees of loss of

cutaneous sensation over the lower parts of the arms and legs. Vibration, muscle and joint sense may also be abolished. A reaction of degeneration is usually present in the affected muscles.

**Differential Diagnosis.** The disease cannot be distinguished with certainty in the early stages from progressive muscular atrophy, which may start in the feet. Later, however, the characteristic distribution of the muscular atrophy establishes the diagnosis. In peripheral neuritis the onset is more acute and the sensory changes are more marked. In the distal type of myopathy of Gowers and Spiller (see p. 600) there are no sensory changes and fibrillation is not present. Claw hand and talipes equino-varus also occur in progressive hypertrophic neuritis (Dejerine-Sottas's disease), but here acute pains occur in the arms and legs, and the peripheral nerves are thickened and palpable.

**Course and Complications.** The course is usually slowly progressive over several years, but arrest of the disease then occurs, as shown by the absence of fibrillary twitching.

**Prognosis.** The disease is not fatal, but the affected muscles do not recover, although an improvement of function often occurs, due to the formation of fibrous tissue.

**Treatment.** The muscles should be massaged. Operations are contra-indicated, but light splints should be worn to help to prevent and correct deformities.

### Progressive Spinal Muscular Atrophy of Infants

#### (Werdnig-Hoffmann Disease)

**Definition.** A disease of infants characterised by progressive weakness of muscles, due to degeneration of their motor neurones.

**Etiology.** The cause is unknown. *Predisposing causes:* 1. The disease may occur in several members of a family. 2. Age: The onset is usually shortly after birth. 3. Sex: Both sexes are affected.

**Pathology.** There is degeneration of the anterior horn cells in the cord supplying the muscles involved, with subsequent degeneration of the anterior nerve roots. There may also be degenerative changes in the bulbar nuclei, in the posterior columns and the pyramidal tracts. The affected muscles are atrophied.

**Clinical Findings.** The weakness of the muscles is usually noticed within 2 months of birth, or it may not show itself until towards the end of the first year. The muscles are flabby, but the nutrition of the infant is generally good. The muscles first affected are those of the trunk and pelvic girdle, the limb muscles proximal to the trunk are subsequently involved, and later the neck muscles; in a terminal stage the muscles supplied by the bulbar nuclei may atrophy. The infant lacks power to support itself and move as a normal child, and if it survives cannot walk at the normal age. Fibrillary twitchings are not usually seen in the affected muscles. There is loss of both superficial and deep reflexes. Sensory changes are sometimes detected.

**Differential Diagnosis.** When the disease is present at birth it

closely resembles amyotonia congenita (see p 601) In the latter, however, there is a tendency towards recovery

**Course and Complications** The disease is usually rapidly progressive Pulmonary complications such as bronchitis or broncho pneumonia may develop

**Prognosis** The disease is fatal in the course of a few weeks or months

**Treatment** There is no known treatment

## THE SPINAL NERVES

### Neuralgia of the Spinal Nerves

**Definition** Pain along the course of a nerve, not associated with a detectable organic lesion

**Etiology** There is little distinction between neuralgia and slight degrees of neuritis and both are probably due to similar causes These include inflammation of the nerve sheath or sensory ganglion, anæmia, cachexia and toxic substances such as alcohol and lead Bacterial and protozoal infections may also be associated with neuralgia, as in influenza syphilis and malaria Reflex neuralgia may occur in association with lesions of such organs as the heart kidneys, ovaries, etc Exposure to cold may also produce an attack **Predisposing causes** 1 Age Usually adults 2 Sex Females predominate

**Clinical Findings** The patient complains of pain in the course of a spinal nerve It often occurs in paroxysms and may tend to recur daily at about the same time

**On Examination** Tenderness may be found at certain points on the course of a nerve or the skin may be hyperæsthetic over the area supplied by the nerve Certain varieties will be mentioned **Cervico occipital neuralgia** Pain occurs in the region of the great occipital nerve over the back of the head The skin may be very sensitive to the touch **Brachial neuralgia** The pain is situated in such sites as the outer part of the shoulder in the area of the circumflex nerve, in the upper arm or forearm or in the region of the median or ulnar nerves **Intercostal neuralgia** The pain is located to an intercostal space, and tender spots may be found at the sites of emergence of the lateral or anterior cutaneous branches (see Fig 23A) **Crural neuralgia** The pain occurs along the front and inner aspect of the thigh (see p 436) **Sciatic neuralgia** Here the pain is felt in the region of the great sciatic nerve or its terminal divisions (see p 436) **Coccydynia** The pain is related to the coccygeal plexus and is felt in the region of the coccyx **Plantar neuralgia** This may be associated with gonorrhœa

**Differential Diagnosis** Every endeavour should be made to exclude a true neuritis especially that due to pressure by a tumour or enlarged glands (see Neuritis p 424) There are no muscular signs, and usually no loss of sensation in neuralgia

**Course and Complications** The course is often prolonged, and there is a tendency to recurrence

**Treatment.** The patient should be thoroughly examined to exclude

any general cause of ill-health, such as anæmia or chronic infection. Local treatment, in the form of heat, is of great value. This may be applied by hot bottles or by diathermy. A sedative mixture containing: Tnc. gelsemii m. 10, sod. bicarb. gr. 15, sod. salicyl. gr. 5, sod. brom. gr. 10, syr. aurant. m. 20, aquam ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  ex aqua t.d.s. will often afford relief. Other sedatives, such as aspirin gr. 10 to 15, may be used to afford relief, or to secure sleep. Opium preparations should not be prescribed for fear of establishing a habit.

### Herpes Zoster

(*Shingles. Zona. The Radiculo-ganglionic Syndrome*)

**Definition.** A painful condition associated with a cutaneous eruption and inflammation of the posterior nerve root ganglia.

**Etiology.** The cause is probably a virus. The so-called symptomatic zoster are examples of the virus infection being associated with certain diseases such as cerebro-spinal meningitis, myelitis, subarachnoid hæmorrhage, spinal carcinoma, or treatment with bismuth, arsenic or gold. In some cases herpes zoster is associated with varicella, the latter disease occurring in a person who is in contact with a patient suffering from herpes. It may also occur in epidemics. An attack of varicella does not afford protection against herpes.

**Pathology.** There is inflammation of one or more posterior root ganglia. The ganglion swells and there is lymphocytic infiltration. The virus can be recovered from the vesicles, but how it reaches the ganglia is not known.

**Clinical Findings.** The patient first complains of pain in the region of a spinal nerve, usually in the chest or trunk, but at times along a limb. In the course of 1 or 2 days the cutaneous eruption appears. This consists of vesicles containing clear fluid on a red background. The temperature is sometimes raised to 99° or 100° F. for a few days at the onset. The cerebro-spinal fluid may show an excess of globulin and of lymphocytes. Certain clinical varieties are described, such as zoster universalis (which may be confused with varicella), ophthalmic zoster, geniculate zoster, and glossopharyngeal and vagal zoster.

**Differential Diagnosis.** In the early stages the pain may be mistaken for that of pleurisy or neuritis. The appearance of the eruption establishes the diagnosis. Herpes febrilis, which occurs in such conditions as the common cold and lobar pneumonia, does not follow a nerve distribution and attacks are liable to recur. The causative virus also differs from that of herpes zoster and it produces encephalitis when injected into rabbits.

**Course and Complications.** The vesicles dry up in about a week, leaving permanent scars. Severe neuralgia may occur as a complication. Lower motor neurone paresis involving the affected or adjacent segments is an occasional complication. Immunity is conferred by one attack.

**Prognosis.** This is good, but in elderly people the post-herpetic neuralgia may be very intractable, causing insomnia.

**Treatment.** The skin lesion should be kept dry, a powder of talc



and zinc oxide being applied, or, as is often more convenient, a gauze and collodion dressing. An intramuscular injection of Pituitrin (ext pit liq B P) 1 ml repeated twice in 48 hours, may cut short an attack and prevent the subsequent appearance of neuralgia. Pain is relieved by such drugs as aspirin gr 10 t i d s and by ultra violet light. For post herpetic neuralgia treatment with X rays to the affected region of the spine or with ionisation or diathermy should be tried and a sedative such as Luminal (phenobarbitonum B P), gr  $\frac{1}{2}$  to 1, taken at night. In very intractable cases chordotomy, or alternatively division of the posterior nerve root between the affected ganglion and the cord together with the roots immediately above and below may be advised. In some cases however these operations fail to relieve the pain.

### Radiculitis

**Definition** Inflammation or degeneration of the spinal nerve roots.

**Etiology** Radiculitis may be due to disease of the vertebræ, such as caries or spondylitis, to meningitis due to syphilis or tuberculosis, to trauma usually produced by traction on a limb, or to the pressure of a cervical rib.

**Clinical Findings** The patient complains of pain, which is often paroxysmal. It is usually located to a band area around the trunk or along an arm or leg.

**On Examination** Hyperæsthesia is generally present in the early stages over the site of the pain. In long standing cases the painful area may become anæsthetic. Bone vibration sense may also be lost in the affected part. Muscular wasting occurs in the groups of muscles supplied by the affected nerve roots. The cerebro spinal fluid may show an excess of lymphocytes.

**Treatment.** This is as for neuritis (see p 425). If the Wassermann reaction is positive a full course of anti syphilitic treatment should be given (see p 388).

### Neuritis

**Definition.** Inflammation or degeneration of a nerve.

**Etiology** Localised neuritis may be due to various causes, such as 1 Trauma 2 Compression by a tumour or enlarged glands 3 Fibrositis, involving the nerve sheath 4 Toxins derived from micro organisms, and possibly from the intestine 5 Division of the nerve results in degeneration. Cold is a predisposing cause. Multiple neuritis is separately considered (see p 429).

**Pathology** In interstitial neuritis inflammatory changes occur in the connective tissue between the nerve fibres. In pressure or section neuritis there is degeneration of the nerve sheath and axis cylinders.

**Clinical Findings** The clinical findings vary in different cases according to which function of the peripheral nerve is chiefly affected, and also according to the nature of the lesion. Thus the symptoms may be chiefly motor, sensory or trophic, but these are usually combined. If the nerve is irritated, as in fibrositic infiltration, the chief

clinical feature is pain of a varying degree. Pain of a burning character is known as "causalgia." The muscles supplied by the nerve are tender, and often cramps, or painful muscular contractions, occur when the limb is at rest. The deep reflexes are often exaggerated. When there is more definite compression neuritis there is muscular wasting of an atrophic type with weakness. The muscles are also tender and there is alteration of cutaneous sensibility. Pain and temperature sensations are abolished over the central part of the affected area, but around this there is a zone in which touch sensation is absent, but pain is felt more acutely than normal. There is usually no reaction of degeneration. If the nerve is completely severed the muscles supplied are paralysed, flaccid and wasted, and the reaction of degeneration is present. The deep reflexes are lost. The muscles are not tender, and there is complete loss of cutaneous sensation. Trophic changes include dryness and smoothness of the skin; a localised sweating and keratosis may occur. There may also be redness or blueness or ulceration of the skin. In *brachial neuritis* pain, numbness or tingling may be felt above the clavicle, in the axilla, arm or hand. In *ulnar neuritis* pain occurs in the arm or forearm on the inner side. The nerve is tender on pressure at the elbow. Sensation may be blunted over the inner part of the hand and the inner one and a half fingers. In *median neuritis* a severe burning pain (causalgia) may be felt in the palm of the hand, with alteration of sensation in the outer part of the hand and outer three and a half digits.

**Treatment.** During the acute stage the patient should rest in bed, the arm being supported on a pillow. A liniment of 1 part of lin. aconit., 2 parts of lin. belladon. and 3 parts of lin. chlorof. (B.P.C.) should be applied daily to the arm, which is then covered with cotton-wool. Electrical treatment is best avoided during the acute stages. Sleep may be secured by the use of hypnotics, such as Medinal (barbitonum sol. B.P.) gr. 5 to 10, and a mixture containing aspirin, such as Acid. acetyl. salicyl. gr. 10, pulv. trag. co. gr. 10, sp. chlorof. m. 7, aq. cari dest. (B.P.C.) ad fl. oz. 1, should be given t.d.s. After the acute stage has subsided light massage should be applied to the arm, and if adhesions have formed in the shoulder joint, they will probably disappear as the arm is used. A search for a septic focus in the mouth, nasopharynx, alimentary or renal tracts should also be made, and, if found, eliminated.

Meralgia paræsthetica is described on p. 137.

### Sciatica

**Definition.** Three conditions are commonly included by the term sciatica. 1. Interstitial neuritis of the sciatic nerve. 2. Symptomatic sciatica in which the nerve is involved by compression or by extension of an inflammatory process. 3. Pain referred along the sciatic nerve which itself is not involved in any pathological process.

**Etiology.** Interstitial neuritis is due to some toxi-infective condition such as focal sepsis, rheumatism, alcoholism, diabetes mellitus, syphilis, etc. Symptomatic sciatica is commonly due to fibrositis spreading

from adjacent muscles and fasciæ to involve the sheath of the sciatic nerve. Other causes include spinal cord tumour, prolapsed intervertebral disc, a pelvic tumour, etc. It does not follow that osteoarthritis of the spine, as revealed by X ray examination, is necessarily the cause of sciatica in an individual case. Pain may be referred reflexly along a healthy sciatic nerve from osteo-arthritis of the hip, sacro iliac strain etc. *Predisposing causes* 1 Age. Usually over 20 years. 2 Sex. Males predominate. 3 Strain of the muscles of the back. 4 Chill, as by sitting on wet grass.

*Pathology* With interstitial neuritis there is inflammation affecting the sheath of the nerve and the connective tissue between the nerve fibrils. Fluid may accumulate in the sheath.

*Clinical Findings* In the common type, which is secondary to fibrositis of the lumbar and gluteal muscles, the onset is usually insidious. The patient is an adult who gives a history of straining his back, as by digging or by lifting. Pain and stiffness are felt across the small of the back. The patient may pay no attention to it and strain his back still further. Indications that the inflammation has spread to the sheath of the sciatic nerve are afforded later, perhaps after 1 or 2 months. There is a sensation of numbness or tingling, often felt in the heel when the foot is first put to the ground after resting. This may quickly pass off and recur from time to time. Further, on sitting a sensation of numbness may be felt in the back of the thigh or in the leg, as if the leg has 'gone to sleep'. Later, pain may be experienced either in the back of the thigh or in the leg. This may also be transient and not give rise to any anxiety. If the disease progresses the pain becomes more acute and more persistent. The patient finds that he cannot sit on any ordinary chair with comfort, the weight is put on the sound side and a soft cushion is welcomed. On lying he is usually free from pain, but twitching or cramps may occur in the calf muscles and disturb sleep. Later, the pain may be more acute and disturb sleep. The patient then usually prefers to lie on his back, with the hip and knee flexed and the ankle plantar flexed. It is very difficult for him to turn over on his face owing to pain. If he lies on his side, it is usually on the affected side because lying on the sound side causes a drag in the gluteal region on the affected side. Walking now becomes difficult, the hip and knee are kept slightly flexed and the heel is not put to the ground. The pain in sciatica can be very acute, of a deep seated burning and boring nature, causing considerable exhaustion.

*On Examination* In a developed case definite signs are present. *Spinal motor system* The affected leg (a) Power. This is usually normal but some weakness of the knee flexors or leg muscles may develop later. (b) Tone. There is flaccidity of the affected muscles. (c) Wasting. This is often present in the calf and thigh and the gluteal fold is much diminished. (d) Contractions. Fibrillation and cramp may occur in the calf muscles.

*Spinal sensory system*: Complete anaesthesia is rarely found, but there may be blunting of sensation on the dorsum or side of the foot or on the outer side of the leg. Tender points may be found along the

course of the sciatic nerve in the back of the thigh, or in the calf. There is pain on stretching the sciatic nerve, when the hip is flexed with the knee extended (Laségue's sign). There is no pain on abducting or externally rotating the hip, with the knee flexed.

*The Reflexes.* The knee-jerk is normal or exaggerated. The ankle-jerk is often diminished or lost. The plantar reflex is normal or absent.

Examination of the back, with the patient lying prone on a cushion placed under the lower part of the abdomen, often reveals the presence of fibrous nodules in the lumbar and gluteal muscles. These are tender on pressure. In cases due to a pelvic tumour, rectal examination may reveal the cause, and this should be carried out in every case of sciatica. A sarcoma of the ilium may be obvious on external examination of the back. An X-ray examination should always be made of the spine and pelvis, including the hip joint, as in this way arthritis may be detected. The urine should also always be tested for glucose. Bilateral sciatica is very suggestive of a pelvic tumour, but it may occur in diabetic or in fibrositic sciatica. In some cases fibrositic sciatica has an acute onset. Sicard distinguishes certain types of sciatica according to the position of the lesion. Special names are applied which are as follows: *Neurodocitis*, here the lesion is situated in the intervertebral bony canal. *Radiculitis*, the lesion affects the posterior nerve root within the theca. *Funiculitis*, the lesion is between the posterior root ganglion and the plexus. *Plexitis*, the lesion is situated in the nerve plexus. *Trunkulitis*, the main nerve trunk is affected. *Neuritis*, the lesion is situated peripheral to the nerve trunk.

Clinically, a high sciatica implies a neurodocitis and funiculitis, a middle sciatica a plexitis, a lesion as low down as the popliteal space a trunkulitis and neuritis, and in a low sciatica the lesion is below the popliteal space. In radiculitis there is usually an excess of lymphocytes in the cerebro-spinal fluid, but this is not the case in funiculitis.

*Differential Diagnosis.* Neuralgia may affect the sciatic nerve. The symptoms are not usually so severe as in sciatic neuritis, and objective signs, such as alteration of sensation, loss of the ankle-jerk and muscular wasting are not generally present. In arthritis of the hip there is some limitation of movement of the joint and the X-ray findings are usually diagnostic. Prolapse of an intervertebral disc is considered on p. 428.

*Course and Complications.* Fibrositic sciatica usually pursues a prolonged course lasting several months or 1 or 2 years, but the ultimate tendency is to recovery. Recurrences undoubtedly occur in the same leg. After a severe attack the sciatic nerve remains a weak spot in the body, and any violent strain may provoke another attack. Permanent muscular wasting and diminution of the ankle-jerk are liable to remain. The course in pressure sciatica is progressive unless the cause is removed.

*Prognosis.* The outlook is good in fibrositic sciatica, but in pressure sciatica the prognosis is generally unfavourable, unless it is due to a simple tumour, which can be removed.

*Treatment.* The sooner the patient takes to his bed the sooner a

fibrositic sciatica will be cured. Constipation must be adequately treated (see p 62). It is often unsuspected by the patient, although actually present, and in some cases treatment by regular doses of magnesium sulphate, gr 60 mane, is sufficient to effect a cure. The teeth should be X-rayed and if any apical infection is present it should be treated. The blood Wassermann reaction should be determined, and if positive a course of potassium iodide and mercury should be given (see p 388). If diabetes mellitus is found, it will be necessary to administer insulin and a correct diet (see p 631). Local treatment consists in the application of heat to the leg, either by hot bottles, or preferably by diathermy. If fibrositic nodules are found in the gluteal muscles they should be massaged daily and in this way they can be dispersed. No violent massage or nerve stretching should be employed, as this only does harm and aggravates the sciatica. In the initial acute stages, if the pain is very severe and prevents sleep, an injection of morphine sulph gr  $\frac{1}{2}$  into the back of the thigh often produces a good and lasting effect. This should not be repeated on more than one occasion, for fear of establishing a habit. Other analgesics such as aspirin gr. 10 to 15 nocte will usually secure sleep. Acupuncture of the nerve sheath is rarely required or beneficial, and is not recommended. High sciatica may be treated by epidural injections if other measures fail. These consist in the injection through the sacro-coccygeal foramen of 20 mls of 1% Novocain (procaine hydrochlor B.P.) solution and 60 mls of normal saline. The patient should lie down for a day or so after the injection. Cases associated with spinal arthritis are sometimes benefited by manipulation.

### Prolapsed Intervertebral Disc (*Herniated Nucleus Pulposus*)

**Definition** Backward displacement of portions of an intervertebral disc

**Etiology** Since 1934 interest has been increasingly directed to a condition of backward displacement of a ruptured intervertebral disc. A history of trauma to the back is given in about 80% of cases. It is claimed to be the cause of sciatic pain in about 3% of all cases diagnosed as sciatica.

**Pathology** There is rupture of the annulus fibrosus of a disc and extrusion of the nucleus pulposus. On rupture portions protrude into the spinal canal and may cause pressure on a nerve root. The discs most frequently affected lie between L 4 and 5 or L 5 and S 1 vertebrae. Less frequently the lesion occurs in the cervical or thoracic region.

**Clinical Findings** The patient is usually an adult about the age of 40 who complains of recurring attacks of pain in the lower part of the back, followed by intractable pain in the buttock, posterior part of the thigh and postero-lateral region of the calf. Tingling or prickling sensations may be felt on the outer side of the foot. *On Examination* There is usually rigidity of the lumbar spine with scoliosis and absence of the normal lordotic curve. Tenderness may be elicited over L 4 or 5 spines and over the sciatic nerve in the thigh. There is pain on stretching

the sciatic nerve. The ankle-jerk is absent in about 50% of cases and there may be diminution of cutaneous sensation on the outer side of the foot. The cerebro-spinal fluid contains a slight excess of protein in about two-thirds of the cases. These symptoms and signs are very suggestive of a prolapsed disc, and injection of 5 mls of Lipiodol or Neo-Hydriol (ol. iodisat. B.P.Add.) between L3 and 4 vertebræ may reveal a deformity of the dural sac shadow. Lipiodol should only be injected in carefully selected cases in which an operation will be performed if the diagnosis is confirmed.

**Treatment.** Doubtless many cases recover with the usual treatment of sciatica, rest in bed and warmth. If, however, the condition proves intractable the protruding portions of the disc may be removed by laminectomy. In some cases the relief of pain is dramatic, in others the post-operative convalescence is slow and the results may be very unsatisfactory. Post-operative complications are not unknown, preventing the patient from returning to work, and deaths have occurred from wound or bladder sepsis directly resulting from the operation.

### Multiple Neuritis

#### (Polyneuritis. Peripheral Neuritis)

**Definition.** Inflammation or degeneration of several peripheral nerves.

**Etiology.** The causes, which are varied, may be grouped as follows :  
 1. Exogenous chemical substances, such as alcohol, arsenic, lead, mercury, copper, phosphorus, silver, ether, carbon-monoxide, barbitone, etc.  
 2. Toxins of micro-organisms, as in diphtheria, enteric fever, influenza, gonorrhœa, syphilis, malaria, leprosy and tuberculosis.  
 3. Deficiency diseases, such as beri-beri or lack of the vitamin B complex.  
 4. Metabolic disturbances, as in diabetes mellitus, gout, anæmia, and carcinoma.  
 5. Acute infective or toxic polyneuritis is thought to be caused by an unknown toxin or virus. *Predisposing causes* : 1. Age : Usually 20 to 40, but children are affected in diphtheria. 2. Sex : Equal incidence. 3. Cold and exposure may predispose. In some cases no cause is discovered.

**Pathology.** There is parenchymatous degeneration of the peripheral nerves, usually in their distal parts. The toxin is probably carried by the blood stream, and appears to have a special affinity for certain nerves, and in some cases for certain nerve fibres in the nerves. Thus in lead poisoning the motor fibres supplying certain arm muscles are especially liable to be affected (see p. 738).

**Clinical Findings.** The onset is usually insidious with prodromal symptoms. Thus the patient may complain of numbness, tingling or pains in the legs or of cramps in the calves. The feet or legs may feel hot or cold. Various clinical types of neuritis exist, according to the toxic agent. Thus in lead neuritis (see p. 738) the motor nerve fibres are affected usually in the arms, but sometimes in the legs or elsewhere. Both arms or legs are involved, the distribution of the lesion being

**symmetrical** The motor lesions are of the lower neurone type, there being a flaccid paralysis of certain muscles, with wasting and loss of the deep reflexes. A reaction of degeneration may be obtained. Often a mixed type of neuritis is present, with motor, sensory and possibly trophic changes. Thus in alcoholic neuritis (see p 713) the legs are chiefly affected. There is pain in the calves or feet with muscular tenderness. Sensory changes may be present with a stocking area of distribution. Hyperæsthesia or anæsthesia of the foot and lower part of the legs may be found. In the early stages the knee-jerks may be exaggerated but later they are lost. In diabetic neuritis trophic changes may be seen such as a perforating ulcer or gangrene of a toe. Other trophic changes found in different types of neuritis include vasomotor disturbances, such as redness of the skin, a smooth glossy skin, absence of hair, increased sweating, œdema, brittleness of the nails and kerato dermia. Achlorhydria is present in some cases of multiple neuritis. In acute infective polyneuritis there is a rapidly spreading paralysis involving the legs, arms and trunk. The VII cranial nerve may also be affected. There is in addition constitutional disturbance as shown by the rise of temperature. The Guillain Barré syndrome is characterised by a diffuse polyneuritis, the cerebro spinal fluid contains an excess of albumin but no cells. Fever may or may not be present and the patient almost always recovers completely.

**Differential Diagnosis** In the motor variety of peripheral neuritis other causes of weakness and wasting of muscles must be considered. *The symmetrical distribution of a lower motor neurone lesion is characteristic of peripheral neuritis.* In anterior poliomyelitis (see p 407) the illness is acute and special groups of muscles are picked out. The temperature is also usually raised. In acute myelitis the paralysis involves the lower part of the body to a certain level, the sphincters are usually affected, which is not the case in peripheral neuritis, and sensory changes extend up to a definite segmental level. Landry's paralysis closely resembles acute infective polyneuritis (see p 410). When ataxia and pains in the limbs are prominent features, tabes dorsalis may be suggested. In the latter, the anæsthetic patches on the trunk and nose, the pupil changes and the examination of the cerebro spinal fluid are diagnostic (see p 390). The trophic type may suggest Raynaud's disease, erythromelalgia, acrocyanosis or syringomyelia (see pp 272, 273, 415). There are usually some motor changes present as well in peripheral neuritis and the stocking and glove distribution of sensory changes is suggestive of peripheral neuritis.

**Course and Complications** The course is usually chronic, the symptoms lasting several months. In acute infective polyneuritis the course is, however, more rapid. Mental changes, such as Korsakow's psychosis (see p 713) are especially liable to occur in association with alcoholic neuritis. Myocardial degeneration may also be present, particularly in diphtheritic neuritis and in beriberi. Permanent contracture of the antagonistic muscles may ensue.

**Prognosis.** This is improved when the diagnosis is made early and the cause removed at once. In acute infective polyneuritis the

outlook is always grave, and death may rapidly result from respiratory or cardiac failure.

**Treatment.** The patient should be put to bed and the affected limbs rested. The cause of the neuritis should, if possible, be removed. Contracture of the unaffected muscles must be prevented by the use of light celluloid splints to keep the affected muscles from being over-stretched. Hot fomentations are useful in relieving pain. In addition it may be necessary to give analgesic drugs such as aspirin or phenazone gr. 10 t.d.s. Special treatment is required for the neuritis due to lead (see p. 739) and diphtheria (see p. 542). Treatment with vitamin B<sub>1</sub> is of little, if any, value in alcoholic neuritis. Electrical treatment by diathermy is of value in some cases for the relief of pain. As the pain subsides the muscles should be gently massaged.

### Tumours of Nerves

**Etiology.** The cause is unknown.

**Pathology.** The tumour may be a neuroma or a fibro-neuroma, usually the latter. Single or multiple tumours may be met with. The true neuroma consists of nerve ganglion cells or nerve fibres, and occurs in the sympathetic system. The fibro-neuroma is composed chiefly of connective tissue, and is attached to nerve trunks. When appearing on cutaneous nerves they are known as molluscum fibrosum, and if in addition there is pigmentation of the skin they constitute von Recklinghausen's disease.

**Clinical Findings.** Nerve tumours may give rise to no symptoms. If attached to the posterior nerve roots, they may cause pain, or muscular weakness if they arise from the anterior nerve roots. When situated under the skin they are usually tender to pressure (*tuberculosa dolorosa*).

**Treatment.** If the tumour is causing symptoms and is accessible, it should be removed surgically.

### The Cervical Plexus and its Branches

**Anatomy.** The cervical plexus is formed from the anterior primary divisions of the first four cervical nerves.

### The Phrenic Nerve

**Anatomy.** The phrenic nerve is formed from C. 3, C. 4 and C. 5 nerves, chiefly from C. 4. It passes down the neck over the scalenus anticus muscle, and after traversing the thorax supplies the diaphragm.

**Etiology of Lesions.** Lesions may be due to : 1. Trauma, such as a wound in the neck. 2. Pressure, as by arthritis or fracture of the spine, enlarged glands, a mediastinal tumour or abscess, or a pericardial effusion. 3. Nervous diseases, such as spinal meningitis, poliomyelitis, hæmorrhage of the cord, Landry's paralysis, or neuritis. 4. Avulsion or crushing, as in the treatment of pulmonary tuberculosis (see p. 156).

**Clinical Findings.** Observation in cases of avulsion of the phrenic nerve shows that usually no abnormal physical signs can be detected.



Thus the movement of the lower part of the chest and of the upper part of the abdomen appears normal. The air entry at the base of the lung on the affected side is often weaker owing to the fibrosis of the lung present in such cases. X ray examination shows the diaphragm on the affected side raised 1 or 2 inches and motionless, or exhibiting paradoxical movement (see p 200). In cases of irritation of the diaphragm by enlarged glands due to malignant disease reversal of diaphragmatic movement may occur on the affected side, the paradoxical movement showing the affected half of the diaphragm to ascend with inspiration, when the diaphragm on the sound side descends. Spasm and flutter of the diaphragm are considered on pp 199, 201. If both phrenic nerves are paralysed there is recession of the upper part of the abdomen with inspiration. If the intercostal muscles are also paralysed death occurs from respiratory failure. The relationship of lesions of the phrenic nerve to massive collapse of the lungs is considered on p 169.

### The Brachial Plexus and its Branches

**Anatomy** The brachial plexus is formed from the anterior primary divisions of the 5th to 8th cervical nerves and from part of the 1st thoracic nerve.

**Etiology of Lesions** The brachial plexus may be affected by various lesions such as 1 Trauma, from a wound or by violent traction of the arm, as occurs in obstetrical paralysis. 2 Pressure from a cervical rib or from a fractured or dislocated clavicle or humerus or a subclavian aneurysm. 3 Neuritis (see p 124).

**Clinical Findings** If the lesion affects the whole of the brachial plexus, the corresponding arm and hand are flaccid and paralysed, and sensation is lost over the arm and hand. The sympathetic supply to the eye may also be involved by the injury to the Th 1 root, and the eye on the same side shows a small pupil with enophthalmos and possibly slight ptosis. Only a portion of the plexus may be damaged. Thus if the C 5 and part of the C 6 roots are injured there is paralysis of the upper arm type (Erb Duchenne). This is the type met with in obstetrical paralysis. The muscles paralysed include the deltoid, biceps, brachialis anticus, and the supinator longus, but the sensory loss is usually slight. If the C 8 and Th 1 roots are injured the resulting paralysis is of the lower arm type (Klumpke). There is paralysis of the small muscles of the hand, and the wrist and finger flexors are often affected. Sensory loss is found on the inner side of the hand and forearm, and there may also be a small pupil and enophthalmos on the same side. Cervical ribs usually cause lesions of the lower arm type. Such a cervical rib is liable to develop from the 7th cervical vertebra when the brachial plexus is pre fixed, the C 4 root joining the plexus and the C 4 and 5 nerve roots being large, whereas the C 8 and Th 1 roots are small. Often there are bilateral cervical ribs, but symptoms are only found on one side, usually the right. They are due to pressure of the rib on the lower segments of the plexus. They do not usually show themselves until the patient grows

up. In some cases in the 1914-18 war, they were first noticed when the patient wore a pack. The symptoms and signs are of the lower arm type, as described above. There may be numbness, tingling or pain on the inner side of the hand or forearm, which is relieved by elevating the hand. There may also be wasting of the small muscles of the hand and a claw hand may develop. The radial pulse may also be weaker on the affected side.

**Differential Diagnosis.** This is established by X-ray examination, but care should be taken to exclude other diseases, causing muscle wasting of the hand, such as syringomyelia and progressive muscular atrophy.

**Treatment.** If the cervical rib is giving rise to definite symptoms, it should be removed surgically.

### The Long Thoracic Nerve

#### *(The Posterior Thoracic Nerve. The Nerve of Bell)*

**Anatomy.** The long thoracic nerve arises from the C. 5, C. 6 and C. 7 nerve roots as they traverse the intervertebral foramina. It passes through the scalenus medius muscle to the axilla and to the outer side of the serratus magnus muscle, which it innervates.

**Etiology of Lesions.** The long thoracic nerve may be affected by :  
1. Trauma, by a wound in the neck or axilla, or by carrying heavy weights on the shoulder, as may occur with porters. 2. Neuritis, in influenza or diphtheria. 3. A cord or cerebral lesion, in progressive muscular atrophy.

**Clinical Findings.** The patient finds that his upper arm and shoulder are weak. Thus there is difficulty in elevating the arm above the level of the horizontal, and in pushing with the arm.

**On Examination :** When the arm is by the side the scapula on the affected side is higher than the other one, and the inferior angle is nearer the spine. If the patient is asked to hold his arms out in front of him and the back is inspected, there is prominence of the vertebral border of the scapula (winging of the scapula) on the affected side.

### The Circumflex Nerve

**Anatomy.** The circumflex nerve arises from the posterior cord of the brachial plexus from the C. 5 and C. 6 nerves. It travels behind the axillary artery, and winds round the surgical neck of the humerus to supply the deltoid and teres minor muscles. It also gives a branch to the shoulder joint, and a cutaneous nerve to the skin of the upper and outer half of the arm.

**Etiology of Lesions.** The circumflex nerve may be affected by :  
1. Trauma, in dislocation or fracture of the shoulder or by the pressure of a crutch. 2. Arthritis of the shoulder. 3. Neuritis, in fibrositis, diabetes mellitus or exposure to cold.

**Clinical Findings.** The patient complains of pain in the region of the shoulder and the outer and upper part of the arm. He may also experience difficulty in raising the arm or rotating it outwards.

**On Examination :** Flattening of the shoulder may be seen, owing to

wasting of the deltoid. There is some blunting of sensation over the upper and outer part of the arm. In long standing cases adhesions may form in the shoulder joint.

### The Musculo spiral Nerve

**Anatomy** The musculo spiral nerve is formed from the posterior divisions of C 5 C 6 C 7 and C 8 nerve roots.

At first the nerve lies behind the axillary artery, and in the upper third of the arm it passes behind the brachial artery on the inner side of the arm. In the middle third of the arm it passes outwards behind the humerus and reaches the outer side of the humerus in the lower third of the arm, entering the forearm by passing in front of the external condyle of the humerus. It then divides into the radial and posterior interosseous nerves. The musculo spiral nerve supplies the triceps, supinator longus and extensor carpi radialis longior muscles. It gives an internal and external cutaneous branch, the former supplying the skin over the upper third of the inner side of the arm, and the latter the skin over the lower third of the outer and back part of the arm, and the upper half of the back of the forearm, and the upper and inner two thirds of the back of the forearm.

**The Radial Nerve** This supplies the skin over the back of the wrist, the outer side and back of the hand, the back of the thumb and outer two and a half fingers.

**The Posterior Interosseous Nerve** This runs round the outer side of the radius to reach the back of the forearm, and travelling deep to the extensor tendons ends on the back of the carpus. It supplies the following muscles: The extensor carpi radialis brevis, supinator brevis, extensor communis digitorum, extensor minimi digiti, extensor carpi ulnaris, extensor ossis metacarpi pollicis, extensor longus pollicis, extensor brevis pollicis and the extensor indicis.

**Etiology of Lesions** The musculo spiral nerve or its branches may be affected by 1 Trauma by a fractured humerus, pressure of a crutch or by pressure from sleeping on the arm when under the influence of alcohol. 2 Neuritis, due to cold or to lead (see p 738).

**Clinical Findings.** Paralysis of the muscles supplied by the musculo spiral nerve causes wrist drop. There is inability to extend the fingers at the metacarpo phalangeal joints or to extend the elbow or supinate the forearm. There is rarely loss or blunting of sensation over the back of the arm, forearm and outer side of the back of the hand.

**Treatment.** The forearm and hand should be supported on a splint, with the wrist and fingers slightly extended.

### The Ulnar Nerve

**Anatomy** The ulnar nerve is formed from the C 8 and Th 1 nerve roots from the inner cord of the brachial plexus. It passes in the axilla between the axillary artery and vein. In the upper half of the arm it lies in front of the triceps muscle internal to the brachial artery. At the elbow it is placed between the internal condyle of

the humerus and the olecranon process of the ulna. In the forearm it lies between the flexor carpi ulnaris and flexor profundus digitorum muscles, and passing in front of the anterior annular ligament into the palm of the hand it divides into a superficial and deep branch. There are no branches in the upper arm. In the forearm the following branches arise: Muscular, to the flexor carpi ulnaris, and inner half of the flexor profundus digitorum muscles. Cutaneous: (a) Palmar, to the palm of the hand and hypothenar eminence; (b) Dorsal, to the back of the wrist and hand, the little finger and the inner half of the ring finger. The superficial terminal branch supplies the skin of the palmar surface of the inner one and a half fingers. The deep terminal branch is distributed to the following muscles: The flexor brevis minimi digiti, the abductor minimi digiti, the opponens minimi digiti, the interossei, the third and fourth lumbricals, the adductor obliquus and transversus pollicis, and the flexor brevis pollicis.

**Etiology of Lesions.** The ulnar nerve may be affected by: 1. Trauma, such as a wound or injury of the arm or elbow. 2. Neuritis, due to cold or to leprosy.

**Clinical Findings.** If the lesion is situated near the elbow the patient notices weakness in flexing the wrist.

**On Examination:** There may be radial deviation and extension at the wrist joint, the fingers are extended at the metacarpo-phalangeal joints, but flexed at the interphalangeal joints, especially the two inner fingers. There is inability to adduct the thumb and the patient cannot separate the fingers. With a lesion at the wrist a claw hand develops, there is wasting of the hypothenar eminence and of the spaces between the metacarpal bones. There is also loss of sensation over the inner one and a half fingers.

**Treatment.** The arm should be rested, and the forearm and hand supported in a splint.

### The Median Nerve

**Anatomy.** The median nerve is formed from the outer and inner cords of the brachial plexus, from the C. 5, C. 6, C. 7, C. 8 and Th. 1 nerve roots.

The median nerve passes down the front of the arm on the outer side of the brachial artery, and in the lower third of the arm crosses in front of the artery to its inner side. It reaches the forearm between the two heads of the pronator radii teres muscle. It runs down the middle of the forearm deep to the superficial muscles and enters the palm on the outer side of the flexor tendons of the fingers. There are no branches in the arm. In the forearm it gives off the anterior interosseous and the palmar cutaneous branches. The anterior interosseous nerve gives branches to the flexor longus pollicis, and the outer half of the flexor profundus digitorum and the pronator quadratus muscles. The palmar cutaneous branch supplies the skin of the palm. The median nerve in the hand supplies the following muscles: The abductor and opponens pollicis, the superficial head of the flexor brevis pollicis, and the two outer lumbricals. The cutaneous branches are distributed to both sides

of the thumb, the radial side of the index finger, and the adjacent sides of the second and third, and third and fourth fingers

**Etiology of Lesions.** The median nerve may be affected by .  
1. Trauma, such as a wound in the arm or forearm. 2. Pressure, as by contractions of the two heads of the pronator radii teres in playing tennis ("tennis elbow"), or continued use of the arm, as in dentistry.

**Clinical Findings.** If the lesion occurs above the elbow, there is weakness in pronating the forearm and in flexing the wrist and the interphalangeal joints. The thumb also cannot be abducted. The hand is deviated to the ulnar side. When the lesion is located at the wrist there is wasting of the thenar eminence and inability to abduct the thumb. Loss of sensation in both lesions is found over the thumb and the two and a half outer fingers. The patient often complains of severe burning pain (causalgia) in the hand.

**Treatment.** The arm should be rested in a sling, and the pain may be relieved by galvanic treatment.

### The Musculo-cutaneous Nerve

**Anatomy.** The musculo cutaneous nerve arises from the outer cord of the brachial plexus, from the C 5 and C 6 nerves.

The musculo cutaneous nerve passes down the arm between the biceps and brachialis anticus muscles to the elbow. It supplies branches to these two muscles and to the coracobrachialis. In the forearm it supplies the skin on the outer and posterior aspect.

**Etiology of Lesions.** The nerve may be injured by trauma or involved in a neuritis.

**Clinical Findings.** The patient has difficulty in flexing the forearm, and sensation may be lost over the outer side and back of the forearm.

### The Lumbo-sacral Plexus and its Branches

**Anatomy.** The lumbo sacral plexus is formed from the anterior primary divisions of the 1st to 5th lumbar and 1st to 5th sacral nerves.

### The Anterior Crural Nerve

**Anatomy.** The anterior crural nerve is derived from the L. 2, L. 3 and L. 4 nerves. It supplies muscular branches to the iliacus, pectineus, sartorius and quadriceps extensor muscles. The sensory branches are the middle and internal cutaneous nerves passing to the anterior and inner side of the thigh, and the internal saphenous nerve supplying the inner side of the leg, foot and big toe.

**Etiology of Lesions.** The anterior crural nerve may be affected by .  
1. Trauma, in fracture of the pelvis or femur, or by wounds in the groin.  
2. Pressure, due to a psoas abscess, a tumour of the pelvis, enlarged inguinal glands, or an aneurysm of the iliac artery.  
3. Arthritis of the spine or hip.  
4. Neuritis due to diabetes mellitus.  
5. Neuralgia associated with chronic constipation.

**Clinical Findings.** An organic lesion of the anterior crural nerve gives rise to weakness of the thigh, the patient complaining that his knee lets him down.

**On Examination :** There is weakness and wasting of the extensors of the knee and slightly of the hip flexors. The affected muscles are flaccid. Tenderness may be found on pressure on the inner side of the thigh. The knee-jerk is diminished or absent, and the quadriceps extensor muscle may show a reaction of degeneration. There may be anæsthesia or paræsthesia over the inner side of the thigh or leg. In crural neuralgia there is pain on the inner side of the thigh or leg and the inner side of the foot. In all cases a rectal examination should be made to exclude a growth, together with an X-ray examination of the pelvis and spine.

**Prognosis and Treatment.** These vary with the cause. In cases due to neuritis the treatment consists in rest, light massage, diathermy and treatment for constipation.

### The Obturator Nerve

**Anatomy.** The obturator nerve is derived from the L. 2, L. 3 and L. 4 nerves. The superficial branch supplies the adductors of the thigh, and the skin over the inner part of the lower two-thirds of the thigh. The deep branch supplies the obturator externus muscle (an external rotator of the thigh) and the knee-joint.

**Etiology of Lesions.** The nerve may be injured in women during labour, or by the pressure of a pelvic tumour or an obturator hernia.

**Clinical Findings.** In obturator neuralgia there is pain over the inner side of the thigh. When the nerve is definitely injured by pressure there is weakness of the thigh adductors, as shown by difficulty in crossing the leg, and weakness in external rotation of the hip may also be detected. There may be blunting of sensation over the lower part of the inner side of the thigh.

**Treatment.** This varies with the cause.

### The External Cutaneous Nerve

**Anatomy.** The external cutaneous nerve is derived from the L. 2 and L. 3 nerves. It enters the thigh close to the anterior superior iliac spine, beneath the outer end of Poupert's ligament. It is a sensory nerve which supplies the skin on the outer side of the thigh and below the great trochanter of the femur.

**Etiology of Lesions.** Lesions may be due to trauma, to pressure in pregnancy, to neuritis, or to local fibrositis in the tunnel of the fascia lata.

**Clinical Findings.** Neuritis of the external cutaneous nerve is known as meralgia paræsthetica. It is generally caused by tension of the overlying fascia. It usually affects obese, middle-aged men. It begins with a sense of numbness over the antero-lateral aspect of one thigh. Later there is burning, tingling and pain, worse on standing or walking. A tender spot may be found at the outer end of Poupert's ligament, where the nerve enters the thigh.

**Treatment.** In some cases it is advisable to excise the nerve to relieve pain.

## The Great Sciatic Nerve

**Anatomy** The great sciatic nerve is derived from the L 4, L 5, S 1, S 2 and S 3 nerves. The nerve, before division, supplies the hamstring muscles which flex the knee (the biceps femoris, semimembranosus, semitendinosus and a portion of the adductor magnus). It divides into the external and internal popliteal nerves. *The external popliteal nerve (peroneal nerve)* This passes down from the lower part of the thigh to the back of the head of the fibula, where it divides into the anterior tibial and musculo-cutaneous nerves. *The anterior tibial nerve* supplies the ankle flexor muscles and the extensor muscles of the toes (i.e., the tibialis anticus, extensor longus digitorum and peroneus tertius muscles). It also gives a cutaneous branch to the skin between the great and second toes. *The musculo-cutaneous nerve* supplies the peroneus longus and brevis muscles (which evert and extend the ankle) and cutaneous branches to the skin over the front of the lower part of the leg, the dorsum of the foot, the inner side of the great toe, and the adjacent sides of the 2nd, 3rd, 4th and 5th toes. *The internal popliteal nerve (tibial nerve)* This is known as the posterior tibial nerve when it enters the back of the leg (at the lower border of the popliteus muscle). It divides at the ankle into the internal and external plantar nerves. It supplies branches to the calf muscles which extend the ankle and flex the toes (i.e., the gastrocnemius, soleus, plantaris, popliteus, tibialis posterior, flexor longus digitorum and flexor longus hallucis), and cutaneous branches to the outer side of the back of the leg, the heel and back of the sole of the foot. *The internal plantar nerve* This supplies muscular branches to the abductor hallucis, flexor brevis hallucis, flexor brevis digitorum and first lumbrical muscles and cutaneous branches to the inner side of the sole of the foot and the plantar surface of the inner three and a half toes. *The external plantar nerve* This supplies the remaining small muscles of the foot, and cutaneous branches to the outer side of the sole of the foot, and the plantar surface of the outer one and a half toes.

**Etiology of Lesions** The great sciatic nerve may be affected by inflammatory changes of the spine or meninges at its origin from the cord, by a spinal tumour or prolapsed intervertebral disc. In the pelvis it may be involved in a fractured pelvis, or by a tumour, or the pregnant uterus. At the sciatic notch or in the thigh it may be injured by a wound or involved by inflammation of its sheath. The external popliteal nerve is liable to injury and it may be affected in lead neuritis. The internal popliteal nerve is rarely affected.

**Clinical Findings** *Lesions in the pelvis or thigh* These result in paralysis of the hamstrings and all the muscles below the knee. There is inability to flex the knee and the foot is dropped. The patient can neither stand on his heel nor on his toes, but he can usually walk. There is loss of the ankle jerk, and anaesthesia is found over the foot and the lower two-thirds of the calf. *Lesions of the external popliteal nerve* The foot is dropped and inverted, the toes are flexed and cannot be extended. The patient can stand on his toes. There is anaesthesia

on the outer half of the front of the leg and on the dorsum of the foot and adjacent sides of the toes. *Lesions of the internal popliteal nerve:* The patient cannot extend his foot and cannot flex the toes. He cannot stand on his toes. There is anæsthesia on the lower third of the outer and back part of the leg, and the sole of the foot and plantar surface of the toes. Later, contracture may occur causing a claw foot.

**Treatment.** This varies with the cause; if due to injury by a wound, nerve suture may be required. In other cases a pelvic tumour may be present, which can be removed by operation. Many cases are due to inflammatory changes in the nerve or its sheath, and the treatment then is that described for sciatica (see p. 427).



## CHAPTER V

### THE URINARY SYSTEM

**Introductory** Clinical examination in diseases of the urinary system implies far more than an examination of the urine. The cardio vascular system, the blood and the tissue fluids are intimately linked with this excretory system. Information will be required on the following points.

**The urine** The colour, reaction, and odour. The amount and specific gravity, by day and night. The consistency and deposit.

**Chemical examination** Proteins, albumin, globulin, albumose, proteose, mucin, and hæmoglobin derivatives, such as methæmoglobin and porphyrin. Bile salts and pigment. Reducing substances, such as glucose, lactose, pentose, creatinin, uric acid and homogentisic acid. Chlorides.

**Microscopical examination** Blood cells (red and white), pus cells, renal or vesical epithelium, casts, crystals, phosphates, uric acid, urates, oxalates, cystine, leucine and tyrosine. In acid urine the following crystalline substances may be found: Uric acid, sodium urate, hippuric acid, calcium oxalate, cystine, xanthine, leucine and tyrosine. In alkaline urine crystals include calcium phosphate (stellar phosphate), ammonium magnesium phosphate, ammonium urate, calcium carbonate, and cholesterol.

**Bacteria**, such as the *Bacterium commune* (B. coli), the *Mycobacterium tuberculosis* (B. tuberculosis), staphylococci and streptococci.

**Ova**, such as those of the bilharzia.

**The blood** Chemical examination. Total protein, albumin and globulin, urea, non protein nitrogen, cholesterol and chlorides. Blood counts. Wassermann reaction.

**The cardio vascular system** The size of the heart, valvular or myocardial lesions, the condition of the aorta and peripheral vessels, the blood pressure and ophthalmoscopic examination.

#### Abnormalities of the Urine

**Amount** The amount of urine passed in the 24 hours by an adult averages 50 oz.—37 by day and 13 by night. This volume may be increased or decreased.

**Polyuria** This is a symptom of many conditions, such as Nervousness, cold, over-drinking, diuretics, such as tea, beer, etc., diseases such as diabetes insipidus, diabetes mellitus, chronic nephritis and hyperpæsis.

**Oliguria and Anuria** Oliguria may be due to various causes, such as Scanty intake of fluids, exercise, sweating, hysteria and fevers. Anuria may result from 1. Suppression, no urine being formed. This may be due to the acute or the terminal stages of nephritis, severe shock and collapse as in cholera, and operations such as passing a catheter. 2. Obstruction. This may be due to stricture, an enlarged prostate, carcinoma of the bladder blocking the orifices of the ureters, calculi in both ureters, a calculus obstructing one ureter, the other

kidney having been removed, or removal of the sole functioning kidney.

**Specific Gravity.** Normally this varies by day and night, being by day 1,018 and by night 1,026. The specific gravity of a single specimen is valueless as a clinical guide, at least a 12 hours specimen is required.

**Colour and Consistency.** The colour may be pale in a low specific gravity urine, as in chronic nephritis, or in a high specific gravity urine, as in diabetes mellitus. The urine may be turbid or show a deposit, due to: Urates, phosphates, mucin, pus, and organisms. Various colours may be noted, such as: Brown, due to bile or to urates; red, owing to blood or its derivatives, to Prontosil Rubrum or Prontosil Soluble, or to Pyridium when the urine is acid; black, due to melanin, carbohic acid, and alkapton; white, from chyle; green, from bile, methylene blue, and carbohic acid; pink, from a deposit of urates; blue, due to methylene blue; orange, from santonin or rhubarb. The urine may be frothy, due to gas.

### Albuminuria

#### (Proteinuria)

The clinical significance of albuminuria cannot be determined accurately by any test for renal function. There are two groups of cases, benign and organic.

**Benign Albuminuria** (functional, physiological, postural, cyclical, orthostatic). This is liable to occur in growing children, especially boys. At one school it was present in 10% of the boys. It is often associated with lordosis. The morning specimen is free from albumin and globulin, but samples collected after the patient has been up and about contain a small quantity of protein up to 1%. In some cases there is more globulin than albumin. Further, the specimen passed at the end of the day usually contains less than that passed after the patient has been up for only an hour or so. It should be noted that the morning specimen may contain a trace of albumin, owing to the continued elimination of albumin for half an hour or so after the patient first goes to bed. This apparent anomaly can be recognised if the bladder is emptied halfway through the night, when the specimen passed on rising will be free from albumin. The urine contains no casts or only an occasional hyaline or granular one, and oxalate crystals may be present. There are no cardio-vascular changes, the blood pressure is normal, there is no oedema, and the renal function tests are normal. The cause of the condition is not known, but the patient usually grows out of it, although it may last in about one-fifth of the cases until the age of 30. It does not lead to subsequent renal disease. Administration of calcium lactate gr. 10 to 15, or of sodium bicarbonate gr. 60 t.d.s. may cause the albuminuria to disappear temporarily. Violent exercise, such as rowing a course, leads to a temporary albuminuria in the majority of adult athletes. Postural albuminuria may also occur after infections, such as scarlet fever, no albumin being passed as long as the patient remains in bed.

**Organic Albuminuria** *Renal causes* Nephritis, nephrosis, passive congestion (cardiac kidney) infarction, tuberculosis, polycystic disease, malignant tumours, amyloid degeneration, papilloma of the renal pelvis, pyelitis, and calculus *Ureteric causes* A stone *Vesical causes* Cystitis, new growths, calculus, and tuberculosis *Urethral causes* Urethritis and prostatitis

Febrile albuminuria may occur in any condition in which there is moderately high pyrexia, and is probably a manifestation of larval nephrosis (see p 451)

### Globulinuria

Albumin and globulin are usually associated in the urine, where there is albumin there is also globulin In organic cases the ratio is usually about 6 parts of albumin to 1 part of globulin, in functional proteinuria the proportions are more nearly equal

### Albumosuria

#### (Proteosuria)

This may appear during the resolution period of lobar pneumonia, in acute yellow atrophy of the liver, during the puerperium, or in chronic suppuration Bence Jones proteose is associated with multiple myelomata (Kahler's disease, see p 608)

### Phosphaturia

The urine is alkaline The phosphates may only be seen on boiling or they constitute a whitish opacity, often noticed at the end of micturition, and mistaken by the male patient for semen In such instances depression, neurasthenia, or wasting may be noted The condition is often associated with cystitis, due to a staphylococcus infection or it may result from overdosage with alkalis in the treatment of peptic ulcer A specimen removed under aseptic conditions should be examined microscopically and the presence of crystals and organisms determined

**Treatment** This consists in giving acid sodium phosphate in doses sufficient to keep the urine acid, as much as gr 60 t.d.s may be required Hexamine gr 10 t.d.s may also be given (as for pyelitis, see p 170) to try and kill the organisms A diet poor in calcium containing substances is recommended, milk and eggs being avoided

### Oxaluria

This is detected on microscopical examination of the urine It is usually of no clinical significance, but in some instances it is associated with recurrent hæmaturia or with pain suggesting renal calculus

**Treatment** Diet Strawberries, rhubarb, tomatoes, beetroot, spinach, cabbage, radishes, cauliflower, eggs and milk, should be avoided as they are rich in oxalates and lime The following substances which contain magnesium salts may be taken Potatoes, bread, meat apples and peas Acid sod phosph gr 30 to 60 t.d.s and mag sulph (gr 60 to 120) should be given in the morning Hard water should not be drunk

## Glycosuria

The following substances which reduce Fehling's solution may be found in the urine: Glucose, lactose, pentose, homogentisic acid, urates, and creatinin. Benedict's solution is only reduced by glucose, lævulose, lactose, pentose and homogentisic acid. Glucose is of the greatest clinical importance and is considered under diabetes (see p. 627).

## Acetonuria

This is met with in acidosis, associated with diabetes mellitus, Von Gierke's disease, prolonged vomiting and starvation (see Acidosis, p. 637).

## Indicanuria

This can only be detected by chemical tests; an excess is indicative of intestinal stasis.

## Hæmaturia

**Definition.** The presence of red blood cells in the urine.

**Etiology.** The causes are very numerous, and may be thus subdivided: 1. *Renal causes*: Nephritis, infarction, tuberculosis, calculus, crystals such as oxalates or the acetyl derivative of Sulphapyridine, congestion, movable kidney, pyelitis, injury, papilloma, neoplasms, and polycystic disease. 2. *Ureteric causes*: Calculus. 3. *Vesical causes*: Calculus, papilloma, carcinoma, tuberculosis, bilharziasis, acute cystitis, arteriosclerosis, and varicose veins. 4. *Urethral causes*: Trauma, urethritis, ulcerating growths, and caruncle. 5. *Prostatic causes*: Enlargement and growths. 6. *Pre-renal causes*: Leukæmia, purpura, scurvy, hæmophilia, malignant varieties of small-pox, and scarlet fever.

**Clinical Findings.** The colour of the urine varies with the amount of blood present; it may be almost black, or present a smoky appearance, or show a dark ring on top. It may also be only faintly tinged. With small amounts of blood there are no naked-eye changes, but the presence of blood cells is detected microscopically. If the blood is intimately mixed with the urine on passing, it is probably derived from the kidneys; if it appears only at the beginning of micturition it probably comes from the urethra, and if it is noticed at the end of micturition the source may be in the bladder. A case of hæmaturia may be investigated on the following lines: General examination of the patient for fevers, purpura and scurvy. Examination of the heart and arteries in cases of nephritis. Examination of the urine: Amount, casts, crystals, cells, organisms, and ova. This will yield suggestive information as to nephritis, oxaluria, pyuria, infections such as tuberculosis, pyelitis, cystitis, and bilharziasis. Examination of the blood: Blood count, blood culture, Wassermann reaction, blood urea and non-protein nitrogen. These tests will be of help in leukæmia, septicæmia, syphilis, and chronic nephrosclerosis. X-ray examination for stone in the kidneys, ureter, or bladder. Cystoscopy and catheterisation of the ureters, and pyclography. This will show whether the blood is coming

from the bladder or from one or both ureters. Blood coming from one kidney may mean a very early stage of chronic nephrosclerosis or an angioma at the apex of a pyramid, in addition to other causes such as tuberculosis, tumours, calculi, etc. Pyelography is of value in indicating the presence of a tumour deforming the pelvis of the kidney or ureter, or a hydronephrosis. Despite all these investigations in some cases the cause of bleeding cannot be determined, and such cases are known as "renal epistaxis" or the essential hæmaturia of Gull. Certain of these cases are due to local purpuric lesions in the kidney or renal tract.

**Treatment.** This is considered under the various causative conditions.

### *Hæmoglobinuria*

**Definition.** The presence of blood pigments, especially methæmoglobin in the urine.

**Etiology.** Hæmoglobinuria results from intravascular hæmolysis. This may be due to 1 Chemical substances, such as potassium chlorate, arsine, muscarine, sulphuramide or quinine. 2 Infective agents such as cause syphilis, malaria (blackwater fever), and yellow fever. 3 Mismatched blood transfusions. 4 Fabism, idiosyncrasy to the broad bean (*vicia faba*). 5 Pregnancy and the puerperium. 6 Extensive burns. 7 Lederer's anaemia. The cause of this is unknown. 8 Spider bites. A case has been recorded in America resulting from the bite of a large brown spider. 9 Paroxysmal hæmoglobinuria results from (a) exposure to cold, (b) muscular exercise or standing in a position of lordosis, (c) hæmolytic anaemia producing nocturnal hæmoglobinuria, (d) paralytic hæmoglobinuria, or paroxysmal myoglobinuria.

Myoglobinuria is a rare disease in man characterised by muscular atrophy and recurrent attacks of myoglobinuria. It is also met with in the crush syndrome (see p. 431). It occurs commonly in horses which are taken out to work after a rest. Myohæmoglobin derived from the muscles appears in the urine.

Spectroscopic examination of the urine reveals the presence and nature of the pigment. In paroxysmal hæmoglobinuria the patient is often a child or young adult suffering from congenital syphilis. The attack is excited by cold and may be induced by placing the extremities in cold water (Rosenbach test). There is a hæmolysis circulating in the blood, which at low temperatures attaches itself to the red cells and causes their lysis. (This is the basis of the Donath Landsteiner reaction.)

**Treatment.** This varies with the cause. To avoid a mismatched blood transfusion adequate care must be taken in blood grouping, and alkalis sufficient to render the urine alkaline, should be given by mouth to the recipient before and after the transfusion. Cold hæmoglobinuria can often be cured by appropriate anti-syphilitic treatment. In other cases daily intravenous injections of ascorbic acid (B.P. Add.) mg. 300 given for several days, has proved successful. The outlook in the nocturnal hæmolytic anaemic group is very grave.

Repeated small blood transfusions should be given, and in no case should splenectomy be performed.

### Porphyrinuria

The term hæmatoporphyrinuria, formerly employed, is incorrect. Hæmatoporphyrin is not a naturally occurring substance. Porphyrinuria may be due to drugs such as Trional or sulphonal, or rarely it is a congenital metabolic error associated with photosensitivity, skin lesions (hydraea aestivale), and pigmentation of bones and teeth. An acute idiopathic type is also described. The symptoms are abdominal pain due to gastric or intestinal spasm, vomiting and constipation, and the urine is brown or red. The patient is often acutely ill and runs the risk of a laparotomy being performed if the significance of the urinary changes are not appreciated. The urine may be of normal colour when passed, darkening on standing to a brown or red shade (port-wine colour). The pigment is detected spectroscopically.

### Pneumaturia

Gas may be passed with the urine when there is a fistula between the bladder and bowel, or an urethral fistula. Occasionally it is due to the presence of gas-forming organisms in the bladder.

### Pyuria

**Definition.** Pus in the urine.

**Etiology.** The pus may come from the kidney, as in pyelonephritis, pyonephrosis, pyelitis, or renal calculus; from the ureter owing to the presence of a calculus; from the bladder in cystitis, new growth, a calculus, or an adherent and perforated diverticulitis or appendix abscess; and from the urethra in urethritis, or in periurethral suppuration such as may result from a prostatic abscess. The urine may be thick, or the pus may be present only in microscopical amounts. The best test for pus is the microscopical one of finding pus cells in the urinary deposit. The treatment is that of the various causative conditions.

### Renal Function Tests

In medical cases, in addition to a general clinical examination, these tests are important for three varieties of problems:

1. Does albuminuria signify nephritis or permanent renal damage?
2. What is the type and severity of nephritis in any given case?
3. In cases of essential hypertension, is there failure of renal function?

The practitioner will require the following tests:

**Examination of the Urine.** The presence of protein, casts and cells. The determination of the relation of proteinuria to posture and exercise. The amount of urine passed by day and by night, and the specific gravity of the day and night specimens.

**Examination of the Blood.** An increase of urea and non-protein

nitrogen is associated with acute glomerulo nephritis and with malignant nephrosclerosis. A considerable increase renders the prognosis more unfavourable suggesting the danger of uræmia.

*The Excreting Power of the Kidney* This can be roughly determined by the urea concentration test and the water elimination test.

*The Blood Urea Clearance Test (Van Slyke)* This shows the volume of blood whose urea content is excreted in one minute's urine. With 'standard clearance' the normal man excretes 1 c.c. urine a minute, containing the urea content of 5½ c.c. of blood. The results can be expressed as a percentage figure, e.g., the kidneys are found to be 60% efficient. The normal figure is from 70% upwards.

*The Injection of Dyes and their Excretion* These tests are only applicable if there is no blood in the urine, and are chiefly of value in determining the separate function of each kidney, the urine being collected by ureteric catheters.

In or practical clinical medicine the most simple and reliable tests are the examination of the urine for volume, specific gravity, protein, casts and cells, the blood chemistry, the blood urea clearance, and the urea concentration and water elimination tests. In normal cases the urine is more concentrated by night, the volume being less and the specific gravity being higher than in the day specimen (see p. 411). The urine also contains no protein, but a few hyaline casts and red corpuscles are of no significance.

The normal blood chemistry figures are

Blood urea	20 to 40 mg per 100 c.c.
Non protein nitrogen .	20 to 40 mg per 100 c.c.
Creatinin	1 to 1.5 mg per 100 c.c.
Cholesterol	180 to 225 mg per 100 c.c.
Chlorides	500 mg per 100 c.c.
Calcium	9 to 11 mg per 100 c.c.
Phosphates	2.5 mg per 100 c.c.
Uric acid	2 to 3.5 mg per 100 c.c.

A blood urea of over 200 is of serious import, indicating grave renal damage. In the urea concentration test, in the first hour's specimen, a figure below 1.5% urea indicates inefficient excretion and in the second hour's specimen a figure below 2% is also considered abnormal. In the water elimination test the patient fasting from the previous night after drinking a pint of water in the morning should excrete 20 oz. of urine in the next 3 hours.

## NEPHRITIS AND NEPHROSIS

### (Bright's Disease)

**Definition** Bright's disease includes inflammatory and degenerative changes of the kidneys. Suppurative renal lesions are excluded. Various types are described and it is difficult to classify. The outlook with both clinical and pathological findings. The

swollen about the face, he is generally pale, and œdema may be present in the legs, over the lumbar region (lumbar pad), over the sternum and in the scrotum. The abdominal and thoracic wall may become œdematous and the swelling spread over the thighs. After the swelling has subsided, linear atrophicæ are sometimes observed (see Fig. 45). Ascites or pleural effusion may develop later. The swelling causes a feeling of stiffness, as if the body were in a leather case, and the patient often prefers to be propped up owing to a certain degree of dyspnoea. There is usually fever at the onset, with a temperature of  $101^{\circ}\text{F}$  to  $103^{\circ}\text{F}$  falling to normal in a week or 10 days. During the acute stage the blood pressure is generally raised for a few days, rarely above 160 or 180 mm Hg in an adult. Cardiovascular system. There is generally no cardiac enlargement which can be detected clinically or radiographically, but the aortic second sound is often accentuated and a tic tac rhythm may be noted. The pulse rate is increased. The lungs. A few scattered rhonchi or some basal rales are often heard. There are usually no retinal changes, but at times there are signs of hypertensive neuro retinopathy, the arteries are narrowed, veins dilated and a few hæmorrhages are noticed or there may be slight œdema of the discs. The subcutaneous œdema fluid is rich in protein (over 1%). The urine. The volume is reduced to about 10 to 20 oz. or there may be anuria for a day or so. The specific gravity is raised (1.025 to 1.035). The colour is dark owing to blood and urates. Protein is present (0.4 to 2%). Microscopically. Hyaline blood and epithelial casts, renal epithelial cells, and some red and white blood cells are seen, but the urine is usually sterile. The blood. Nitrogen retention often occurs during the acute stage. The protein and cholesterol contents are normal. The urea concentration test should not be performed owing to the strain it places on the kidney. The water elimination test shows low values.

**Differential Diagnosis.** This usually presents little difficulty, the signs and symptoms being characteristic. Focal nephritis is excluded by the presence of œdema, the hypertension and the blood nitrogen retention. In some cases the disease is a reactivation of an old infection when cardiac hypertrophy and arteriosclerosis may be expected. The presence of fatty casts in the urine is also suggestive of old standing disease. Other causes of hæmaturia (see p. 443) must be considered but these are not usually associated with œdema, except in heart failure, where the cardiac condition is obvious.

**Course and Complications.** Acute nephritis is often a self limited disease, after a few days the urinary output suddenly increases to about 60 to 80 oz. a day, the amount of blood excreted diminishes, and the blood pressure falls to normal. The œdema may, however, persist for several weeks, or the condition merge into one of subacute or chronic nephritis (see p. 453). Uræmia, hypertensive encephalopathy, myocardial failure, pericarditis, peritonitis, and acute œdema of the lungs or larynx are of serious import. Bronchitis is not usually severe.

**Prognosis.** Death is uncommon, but may result from one of the complications mentioned above. Cases due to scarlet fever often make



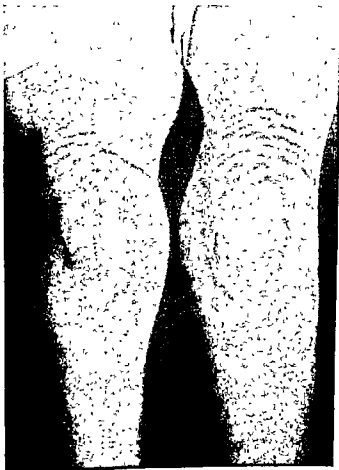


FIG. 45. Linear atrophicae on knees following acute nephritis.

a complete recovery, but there is always the danger of permanent renal damage.

**Treatment. Prophylactic.** In scarlet fever the use of alkalis and of antitoxin may diminish the liability to nephritis. Statistics do not indicate that eradication of septic tonsils in children prevents the incidence of nephritis; in fact it may predispose to it.

**Curative.** The patient should be in bed between blankets, and in flannel pyjamas. The temperature of the room should be maintained at 60° F. to 65° F. Diet: The ordinary "milk diet" should not be given, as this contains about 65 G. of protein, and is rich in salts. The diet should be as follows: *Diet 1*: Days 1 to 3 or 4. The total fluid should not be more than 1 pint in 24 hours, made up of dextrose orangeade (dextrose 8 oz., the juice of one orange and water 1 pint), water, barley water, imperial drink (acid pot. tart. gr. 60, sugar q.s., the peel of half a lemon, and water to 1 pint). A little toffee and raw apple may also be taken. When the urinary output increases and the hæmaturia diminishes, *Diet 2* is given. This is milk,  $\frac{1}{2}$  a pint, diluted with water or soda water, half and half. Barley water or dextrose orangeade, 1 pint. Toffee. Four thin slices of bread and butter. One small cup of Benger's food. Cream,  $\frac{1}{2}$  oz. Weak tea and sugar. In another 3 or 4 days, if the patient improves, the diet may be gradually increased by the addition of the following substances: Milk up to 2 pints in all. Bread and milk, sugar, toast, honey, potatoes, and green vegetables. Then add gradually milk pudding with no eggs, suet pudding, porridge, fruit such as bananas, steamed fish, chicken, boiled mutton, and an egg once a week. All meat and vegetable extracts which are rich in salts and nitrogenous bodies must be avoided, also coffee, alcohol, bacon, ham, cheese, pickles, salt, and red meats. No salt should be used in cooking or taken with food while any œdema is present.

The bowels should be opened daily with salts such as magnesium or sodium sulphate in doses sufficient to produce a loose motion (usually gr. 60 to 120). Pulv. jalap. co. is best avoided owing to its irritant action. If there is œdema, sweating may be induced by hot packs or by a hot air bath (using an electric cradle, the temperature being raised to 120° F. for 20 minutes). Care must be taken not to exhaust the patient, the pulse being watched all the time. It is usually wiser not to give pilocarpine to induce sweating, as œdema of the lungs may result. Hot applications should be placed over the loins to relieve aching. Irritating diuretics should be avoided, but a citrate mixture is useful, such as Pot. cit. gr. 20, pot. acetat. gr. 20, acid pot. tart. gr. 20, liq. ammon. acetat. m. 60, aq. chlorof. ad fl. oz. 1. Fl. oz. 1 should be given during the early stages sufficiently frequently to render alkaline every specimen of urine passed. The treatment of convulsive (hypertensive) uræmia is described on p. 466.

**Convalescence.** The patient must be kept in bed until all blood, albumin and casts have disappeared from the urine, the œdema has gone, and the temperature is normal. The associated anæmia is benefited by an iron mixture such as Basham's mixture, i.e., Pot. cit. gr. 10, liq. ferri acetat. m. 15 (B.P. 1893), liq. ammon. acetat. m. 120,

aq camphor ad fl oz, 1 11 oz 1 tds p.c. A bitter tonic such as *Enc nuc vom* m 10, *sod bicarb* gr 10, *sp chlorof* m 7, *infus gent co rec* ad fl oz 1 11 oz 1 tds, can be given before meals to improve the appetite. The eradication of septic foci, especially in teeth and tonsils is usually advised but there is no definite evidence that tonsillectomy is of any value in curing nephritis or preventing it from progressing to a chronic stage.

### Focal Nephritis

**Definition** A condition characterised by hæmorrhages into certain glomeruli of the kidney.

**Etiology** Focal nephritis is due to bacterial emboli, usually streptococcal. It occurs in association with such acute infections as tonsillitis, otitis media, erysipelas, puerperal fever, pneumonia, scarlet fever (during the first week) influenza meningococcal meningitis, etc. At times it is due to chemical substances, such as mercury, phosphorus, arsenic, cantharides, etc. In subacute infective endocarditis a condition of multiple glomerular embolisation often occurs.

**Pathology** There is hæmorrhage into certain glomeruli, and there may be cloudy swelling of the epithelium of the tubules. In subacute infective endocarditis multiple infarcts occur in the glomerular vessels.

**Clinical Findings** The patient is a child or an adult who, during the acute stage of one of the illnesses mentioned above, passes blood in the urine. The urine may be diminished in volume, containing blood, albumin and hyaline granular and blood casts. Streptococci can often be found in the urine. There is no œdema, no increase of blood pressure and no nitrogen retention in the blood.

**Differential Diagnosis** Focal nephritis is differentiated from acute diffuse glomerulo-nephritis by its onset during the acute stages of an infection and the absence of œdema and hypertension.

**Course and Complications** The urinary changes usually rapidly disappear, rarely albuminuria persists for some time.

**Treatment** No special treatment is required for the renal lesion. If there is oliguria, the fluid intake should be restricted to 1 to 2 pints until diuresis ensues. After recovery focal sepsis in the tonsils or teeth should be treated.

### Acute Interstitial Nephritis

**Etiology** Acute interstitial nephritis occurs in association with diphtheria, scarlet fever, septicæmia and streptococcal tonsillitis.

**Pathology** Areas of infiltration with red and white blood cells and plasma cells are seen in the cortex between the tubules and glomeruli.

**Clinical Findings** It is impossible to diagnose this condition during life. In some cases there is oliguria with slight albuminuria. Usually there is no hæmaturia, no œdema, and no hypertension.

### Acute Nephrosis (Toxic Nephritis)

**Definition.** Acute degeneration of the renal parenchyma.

**Etiology.** There are three chief varieties described: 1. *Larval*, as in febrile albuminuria, and albuminuria associated with diabetes mellitus, Graves' disease, jaundice, pernicious anæmia, and chemical substances such as mercury (in small amounts) and phosphorus. 2. *Necrotising*. This may be due to chemical substances such as mercury or bismuth, or be caused by cholera, or by high intestinal or pyloric obstruction. 3. *Hæmoglobinuric*. This may result from various causes such as a mismatched blood transfusion, paroxysmal hæmoglobinuria, blackwater fever, mushroom poisoning, and the crush syndrome.

**Pathology.** In larval nephrosis there is probably cloudy swelling of the tubular epithelium. In necrotising nephrosis there is necrosis of the renal tubular epithelium, but the vessels are normal. The tubules may be obstructed by the necrotic cells which have been shed, and which may be calcified.

**Clinical Findings.** 1. *Larval nephrosis*. Small amounts of albumin appear in the urine, and hyaline casts or a few granular casts may be present. There is no œdema, no hypertension, and no nitrogen retention in the blood. 2. *Necrotising nephrosis*. The urine: There is oliguria or anuria. The albumin is usually less than 1%. The deposit contains hyaline, granular and epithelial casts, but no doubly refractile lipoids. There may be a few red and white cells. The blood: There is marked nitrogen retention, and fall in the alkali reserve. 3. *Hæmoglobinuric nephrosis*. The reader is referred to the articles on Hæmoglobinuria, Blackwater Fever, and the Crush Syndrome (see pp. 444, 682).

**Course and Complications.** Larval nephrosis usually rapidly improves. Necrotising nephrosis is a serious condition; if there is anuria the patient may rapidly die from uræmia. If the patient recovers there is no permanent damage left in the kidney.

**Treatment.** This depends upon the cause; no treatment is required for larval nephrosis. The treatment for necrotising nephrosis is usually that necessary for acute mercurial poisoning.

### The Crush or Compression Syndrome

**Etiology.** This syndrome, which was described in Germany in the 1914-18 war, has attracted attention in England as the result of injuries sustained in air raids. It is liable to occur when a limb is crushed by fallen masonry, etc. Accidents in civil life, such as a fractured pelvis with central dislocation of the hip causing obstruction of the external iliac vessels, may result in the syndrome, without there being apparent muscular damage. It has also been described after a difficult labour.

**Pathology.** There is œdema of the compressed limb, and, as the result of ischaemia, the muscles may show necrosis and at times hæmorrhage. Owing to the œdema, the tension is increased within the fascial compartments. The essential renal lesion appears to be an acute

**hæmoglobinuric nephrosis** There is degeneration of epithelium, with later some regenerative changes in the ascending portion of the loops of Henle and in the second convoluted tubules. Brown casts of desquamated epithelium are seen in the second convoluted and the collecting tubules. Some of the tubules are ruptured. The pigment is myohæmoglobin. The generally accepted view is that the renal lesions are due to myohæmoglobin, derived from the compressed or crushed muscles, which enters the circulation when the compression is released. Potassium salts also escape from the muscles into the circulation. Renal deposition of the myohæmoglobin may be favoured by a fall in the alkali reserve of the blood. Alternatively, it has been suggested that the renal lesions are due to the loss of substances from the blood into the œdema fluid of the compressed limb.

**Clinical Findings** The patient, on admission to hospital, may or may not be suffering from shock. In the former case he is pale, sweating and clammy, the blood pressure is low, the pulse feeble, and the Hb % increased. The affected limb is swollen and œdematous, there may be areas of cutaneous erythema and vesicle formation. Pulsation may or may not be present in the peripheral arteries. The muscles feel firm, the power of moving the limb is restricted, and paræsthesia may be present. Signs of renal failure are noted about the first or second day. Early signs of failure are oliguria or even anuria, and the appearance of blood and albumin in the urine. Brown casts of epithelial cells are present and the pigment is shown spectroscopically to be myohæmoglobin. The urinary urea and chloride figures fall, the blood urea and potassium rise, and the alkali reserve falls. The blood pressure tends to rise and remain above normal if the patient is transfused with serum or plasma. The terminal uræmic stages comprise vomiting, excessive thirst, coma, and death. Cardiac irregularities may occur, and it has been suggested that they are due to potassium retained in the blood.

**Prognosis** Recovery occurs in about one-third of the cases. The length of time that the limb has been compressed appears to bear no direct relationship to the prognosis. A fall in the blood urea is a favourable sign. In fatal cases death usually occurs between the fifth and eighth days.

**Treatment.** Diuresis should be provoked by the administration of large quantities of fluid by mouth or by a rectal drip of 30 oz of 2% sod bicarb solution. Alternatively, 20 oz of isotonic sod. sulphat solution (1.12%) may be given intravenously. Alkalinisation of the urine can also be effected by giving by mouth sod bicarb and sod cit.  $\bar{a}\bar{a}$  gr 150 daily. Whole blood transfusions do not appear of value and may be harmful, but plasma or serum transfusions may be beneficial. The best way of dealing with the compressed limb is a matter of dispute. Some advise that a tourniquet should be applied as soon as the weight is removed from the limb, and that decompression should subsequently be slowly effected, when a good outflow of alkaline urine has been obtained. Others recommend that the limb should be packed in ice, or that incisions should be made to release the œdema fluid.

Primary amputation of the limb with the idea of saving the kidneys has resulted in the loss of both life and limb. The alternative view, based on the theory that the blood has lost vital principles into the œdema fluid, is that the compression should be rapidly relieved, the limb bandaged from below upwards and intermittent pressure, up to 60 mm. Hg., applied over the œdematous limb, using a specially large sphygmomanometer cuff.

### The Kidney of Pregnancy

The kidney of pregnancy must be differentiated from an exacerbation of chronic glomerulo-nephritis which, when present, usually shows itself during the first few months of pregnancy (see p. 454). The kidney of pregnancy resembles a nephrosis in that the renal changes consist in a degeneration of tubular epithelium. The cause is unknown. It is especially liable to develop in the second half of pregnancy, and in elderly primiparæ. It differs clinically from a typical nephrosis in that the blood pressure often rises considerably and convulsive attacks (eclampsia) are liable to occur. The renal function tests are usually normal, including the blood urea and non-protein nitrogen, and urea concentration test.

### Subacute Nephritis

*(Large White Kidney. Large Red Kidney. Large Mottled Kidney.)*

**Etiology.** Subacute nephritis may be a sequel of acute diffuse glomerulo-nephritis or a recrudescence of activity in chronic diffuse glomerulo-nephritis.

**Pathology.** Macroscopically: The kidney is enlarged, the capsule strips easily, and the surface veins are evident. The kidney may be pale, mottled, or red. The cortex is enlarged and the pyramids are dark. Microscopically: The convoluted tubules are chiefly affected, with cloudy swelling, fatty degeneration, and desquamation of the lining cells blocking the tubules. There may be some swelling of the glomeruli, and œdema or proliferation of the tissue in the interstitial structures.

**Clinical Findings.** The patient is often a young adult, who gives a history of acute nephritis, either recently or some years before.

**On Examination:** There is general œdema, the heart may be enlarged, and the blood pressure raised. The urine: The volume is diminished and the specific gravity low. The protein varies from a trace to about 2%. The deposit may contain red cells, leucocytes, epithelial cells, and blood, granular, epithelial and hyaline casts. The blood: The urea and non-protein nitrogen tend to rise, but the cholesterol and protein are normal.

**Differential Diagnosis.** Subacute nephritis must be differentiated from cardiac œdema with albuminuria, from chronic nephrosis, and chronic glomerulo-nephritis (chronic parenchymatous nephritis). The onset of œdema in the legs, the signs of a failing heart, and the absence of cholesterol retention in the blood and of fatty casts in the urine, are

guiding marks in the diagnosis of cardiac œdema, but the differentiation of primary cardiac and primary renal disease is often very difficult

**Course and Complications.** The patient may remain in a waterlogged condition until complications such as pericarditis, ulceration of the colon, bronchopneumonia, or uræmia usher in the fatal issue. In other cases the condition responds to treatment the œdema disappears, and the urinary output increases. In still other instances secondary fibrotic changes occur in the kidneys (small white kidney) with cardiac hypertrophy and increased blood pressure, and the œdema may diminish.

**Prognosis** This is very unfavourable in cases which do not respond to treatment, and in which the œdema does not disappear. Albuminuric retinitis is also an unfavourable sign.

**Treatment** This is as for chronic diffuse glomerulo nephritis with œdema (see below)

### Chronic Diffuse Glomerulo-nephritis

#### (Nephrotic Nephritis)

**Etiology** This form of nephritis is considered to be a sequela of acute diffuse glomerulo nephritis, but in some cases the initial acute attack may have passed unnoticed. It is more likely to follow septic infections than scarlet fever, probably because the former tend to recur, whereas in the latter there is complete recovery from the infective agent. There are two main pathological types of this disease. 1 The large white kidney which may be found up to 3 or 4 years after the acute nephritis. 2 The secondary contracted kidney (small white kidney) which results from secondary fibrosis.

#### The Œdematous Type

##### (Large White Kidney)

**Pathology.** The appearances of the large white kidney resemble those described for subacute nephritis (see p. 453).

**Clinical Findings** In the early stages the clinical picture closely resembles either that of subacute nephritis (see p. 453) or of chronic nephrosis (see p. 458). The characteristic features are the presence of œdema, rise of blood pressure, and retention of nitrogen in the blood. The urine is either normal in amount or slightly diminished. Sp. Gr. 1.020 to 1.026. Protein, 0.5 to 1%. Deposit, epithelial cells, a few red and white blood cells and some hyaline and granular casts. The urea concentration and water elimination tests usually indicate deficient renal function. The œdema fluid is rich in protein, over 1%.

**Differential Diagnosis** In some cases it is impossible to differentiate chronic glomerulo nephritis from chronic nephrosis. The history of previous acute nephritis, a raised blood pressure, and the presence of more than a few red blood cells in the urine are characteristic of glomerulo nephritis.

**Course and Complications** The course is usually slowly progressive. Exacerbations characterised by the presence of blood in the urine may

occur. The patient may die from renal failure or from intercurrent disease before a secondary contracted kidney develops. The œdema may increase, owing to the occurrence of nephrotic changes in the kidney or of cardiac failure. Complications also include pericarditis, ulceration of the colon, and bronchopneumonia.

**Prognosis.** This is very unfavourable in cases which do not respond to treatment and in which the œdema does not disappear. It is also unfavourable if the disease progresses to the stage of secondary contracted kidney.

**Treatment.** The patient should be kept warm in bed during the œdematous stage. In order to try and abolish the œdema various measures may be instituted: *Dietetic.* A Karel salt-poor diet may be given. This consists of milk, 7 oz., 4 times a day, at 8 a.m., 12 noon, 4 p.m. and 8 p.m. On the eighth day one softly-cooked egg and one slice of toast (1 oz.) are added at 10 a.m. On the ninth day another slice of toast is given at 4.30 p.m. and 2 oz. of asparagus, celery, cauliflower, or carrots. Two teaspoonfuls of cornflour and potato are added to the noon milk feed to form vegetable soup. On the tenth to twelfth days one egg, 1 oz. of rice (weighed uncooked) and 2 oz. of the vegetables are added. If the patient does not respond to this salt-poor diet a moderate protein diet containing about 60 to 80 G., with a calorie value of about 2,200, should now be tried.

*Moderate Protein Diet.* Breakfast: Egg, 1; bread, 3 oz., or toast, 2 oz.; butter,  $\frac{1}{2}$  oz.; milk, 4 oz.; marmalade or honey,  $\frac{1}{2}$  oz.; tea, 5 oz. Lunch: White fish (cooked), 3 oz.; potato, 4 oz.; green vegetables, 2 oz.; stewed fruit, 2 oz.; butter,  $\frac{1}{2}$  oz.; bread, 3 oz., or toast, 2 oz. Tea: Bread, 2 oz.; butter,  $\frac{1}{2}$  oz.; jam,  $\frac{1}{2}$  oz.; milk, 2 oz.; tea, 5 oz. Dinner: Chicken or lean meat (cooked), 3 oz.; potato, 4 oz.; green vegetables, 2 oz.; fruit, 4 oz.; bread, 2 oz.; butter,  $\frac{1}{2}$  oz. Sugar for sweetening during day, 2 oz.

Bread made without salt should be used. No salt must be added in cooking or subsequently.

*Diuretics and Diaphoretics.* The following drugs may be tried: Urea 15 G. (gr. 225) in 5 oz. of water 3 times a day, flavoured with syr. aurantii. Theophylline and sodium acetate gr. 2 in a cup of tea 3 times a day for 3 or 4 days. Guy's pill (digitalis gr. 1, pil. hydrarg. gr. 1, and squills gr. 1) 1 pill 3 times a day. Potassium chloride, 5 G. daily, sprinkled on the food, combined with a salt-poor diet. Potassium salts may cause vomiting and diarrhœa if there is much impairment of renal function. Salyrgan (mersalylum B.P.) or Novurit. These should not be given if there is hæmaturia. The method of administration is described on p. 228. Hot air baths to encourage sweating may also be employed (see p. 449).

*Drainage of Fluid.* The peritoneal or pleural cavity may be aspirated, or the legs drained by small incisions, which are kept covered with sterile gauze, while the patient sits up in a chair for 1 to 3 days.

*Alkali Treatment.* Here the diet is not salt-poor but is of the ordinary mixed type containing a moderate amount of protein (60 to 80 G., see above).



Alkalis are given in increasing doses, beginning with Pot cit, pot bicarb, sod cit and sod bicarb, aa gr 15, sp chlorof m 7 and aq menth pip dest to the ounce, 3 times a day. This is increased day by day until the patient may be taking a total amount of gr 960 in all in a day. The œdema usually increases at first, until a sudden diuresis occurs with loss of tissue fluid. This method requires laboratory control of the alkali reserve in the blood, as with such massive doses there is danger of tetany or diarrhœa.

Massage is a useful adjunct to treatment, especially when applied to the extremities. During convalescence an iron mixture should be given if there is anæmia such as Basham's mixture, *i.e.*, Pot cit gr 10, liq ferri acetat (B P 1898) m 15, liq ammon acetat m 120, aq camphor ad fl oz 1. Fl. oz 1 tds. The bowels should be kept open daily with a dose of white mixture.

### The Non-œdematous Type

#### (Secondary Contracted Kidney Small White Kidney)

**Pathology** The kidney is small, firm and irregular. The capsule is adherent and the cortex narrow. There is interstitial replacement fibrosis and arteriolosclerosis. Many of the glomeruli are destroyed and the tubules may be dilated their epithelium being flattened. Areas of healthy kidney tissue may be seen adjacent to the diseased portions.

**Clinical Findings** The patient may complain of headache, dyspnœa, weakness, frequency of micturition especially at night, and symptoms of chronic uræmia such as nausea, vomiting, or diarrhœa, and loss of weight.

**On Examination** The urine. The volume is usually increased, relatively more being passed by night. Sp Gr 1,005 to 1,012. A trace of albumin may be present. Deposit, a few epithelial and blood cells and an occasional hyaline or granular cast. The blood. The urea and non protein nitrogen are raised, the cholesterol and protein normal. The heart. There is a tendency to hypertrophy of the left ventricle and the blood pressure is raised. The arteries are thickened. Ophthalmoscopic examination. Retinal hæmorrhages may be seen with tortuous arteries passing over constricted veins. In children the disease may occur insidiously and give rise to a condition of renal infantilism (also known as renal dwarfism or renal rickets) the growth being stunted, the bones deformed and the secondary sexual characteristics in abeyance.

**Differential Diagnosis** It may be impossible to differentiate this condition from essential hypertension (see p. 260).

**Course and Complications** The course is progressive with gradual failure of renal function. Complications due to hypertension such as convulsions or cerebral hæmorrhage, may occur.

**Prognosis** This is very unfavourable. The patient usually dies from uræmia within two years of the appearance of retinal changes or of a marked rise in the blood nitrogen, or death may be due to

heart failure. Pregnancy affects the condition adversely and should be prevented or terminated.

**Treatment.** This is as described for nephrosclerosis (see p. 462).

### Chronic Nephrosis

#### (*Lipoid Nephrosis*)

**Etiology.** The cause of chronic nephrosis is usually unknown. In some cases it has been suggested that it is secondary to a healed chronic diffuse glomerulo-nephritis. Secondary syphilis at times produces chronic nephrosis (formerly called acute syphilitic nephritis). Rarely it results from diphtheria and CO poisoning.

**Pathology.** Volhard and Fahr considered nephrosis to be a primary degeneration of the renal tubular epithelium, whereas Epstein believed that it is a metabolic disorder, due to a toxæmia and associated with hypothyroidism, the tissue fats being mobilised in the blood, and serum albumin being excreted as a foreign protein. It is now believed that there is increased glomerular permeability to protein and lipoids. The smaller albumin molecules pass through, rather than the larger globulin ones. The function of the tubules is not impaired, as shown by the high Sp. Gr. and ammonia content of the urine. The absence of a high blood pressure indicates that the renal circulation is not affected. The kidneys are either normal in size or enlarged. The capsule strips readily, the cortex is enlarged and shows yellow streaks. Microscopically there is lipoidosis of the tubular epithelium.

**Pathology of Renal Œdema.** Various theories have been proposed to account for this. 1. *Protein drain.* The colloid osmotic pressure of the blood is lowered by loss of protein in the urine, and hence fluid passes from the blood into the tissue spaces. The protein lost from the plasma is albumin rather than globulin, possibly owing to the smaller size of its molecule. Œdema occurs when the plasma protein falls below 5%. This is the chief cause of nephrotic œdema. A fall in plasma protein may occur in conditions such as ascites or after a severe internal hæmorrhage, in which there is œdema without albuminuria or evidence of renal damage. This is known as extrarenal plasma-phoresis. In cachetic and nutritional œdema there is deficient protein intake and a low plasma protein content. Deficiency of vitamin B<sub>1</sub> may also play a part. In nephrosis, therefore, the kidney is presumably damaged first and the albumin drain from the plasma and the œdema are secondary phenomena. 2. *Acidosis.* This is often present and may affect the permeability of the capillary endothelial cells. In some cases the œdema disappears and the urinary output rises when the alkalinity of the blood is restored by sodium bicarbonate. On the other hand, the administration of ammon. chlorid. may provoke diuresis. 3. *Salt retention.* This is usually present, and a reduction of salt in the diet often relieves the œdema. It is the presence of the Na ion and not of the Cl ion which favours water retention. Potassium chloride does not have this effect. Sodium is present in interstitial, and potassium in intracellular tissues. 4. *Lipoid tissue drain.* The blood

cholesterol is raised in the nephrotic type the tissue cells presumably being starved of lipid which may result in their attracting water. There is usually a lowered basal metabolic rate which accompanies a fall in the rate of water transit through the body. 5 *Hydræmia* Inability of the kidney to excrete water does not of itself appear sufficient to explain the dropsy. With anuria lasting for two weeks there is no œdema. 6 *Circulating toxins and anoxia* These damage the capillary endothelium and allow the passage out of proteins. 7 *Raised capillary blood pressure* This is a factor in acute glomerulo-nephritic and in cardiac œdema.

The œdema of acute diffuse glomerulo-nephritis cannot be due to protein drain. The œdema fluid is rich in protein 0.6 to 1% and the plasma proteins are normal. The blood volume remains the same but the urinary output is very low. There is perhaps a lessened power of the tissue cells to retain water owing to an alteration in their electrolytes. As the tissues can hold an excess of about 6000 c.c. of fluid before œdema is clinically manifest, it appears improbable that in the rapidly developing œdema of acute nephritis they are saturated to this extent. It is probable that the cutaneous capillaries are simultaneously damaged with those of the kidney by the same noxious agent which causes the acute nephritis. A further factor is rise in the capillary blood pressure.

*Summary* There are therefore three main elements concerned with the production of renal œdema. 1 Nephrotic 2 Nephritic 3 Cardiac.

*Clinical Findings* The patient is usually a child or young adult. The disease begins insidiously with languor, headache, pallor, swelling of the face, legs, scrotum or abdomen, and possibly some nausea or diarrhoea.

*On Examination* There is a fairly generalised œdema and ascites and hydrothorax may also be present. The face is pale but often there is no real anæmia and no cardiovascular changes develop. The blood pressure is not raised. Retinal changes do not occur. The œdema fluid contains little protein less than 0.1%. The basal metabolic rate is low. The urine. Volume 20 to 30 oz. Sp. Gr. 1.020 to 1.040. Acid (pH 5.4). Protein 1 to 6%, chiefly albumin. There may be a trace of sugar. Deposit leucocytes and epithelial cells and occasionally a red cell. Hyaline epithelial granular and fatty casts and doubly refractile lipid particles. Urea concentration test normal. Water elimination test shows low excretion. This is due to pre renal deviation and not to failure of renal function. The blood. Protein about 4% (normal being 7.4%) of which 89% is globulin (normal being 37% globulin). There is retention of cholesterol (300 to 2300 mg. per 100 c.c., the normal being 180), and lipæmia. The urea and non protein nitrogen are normal.

Such a case as this conforms to the type described as lipid nephrosis, the renal changes being degenerative rather than inflammatory, and it is differentiated from the nephrotic type of chronic glomerulo-nephritis by the history and the tendency to hypertension, and from amyloid

nephrosis by the associated suppurative, syphilitic or tuberculous lesions.

**Course and Complications.** Complete recovery is said to be the rule if proper treatment is given; but this is not always the case. The course is variable and death may result either from failure of renal function, with gradual increase of blood nitrogen and eventually uræmia, or from complications such as pneumococcal peritonitis and erysipelas.

**Treatment.** The Wassermann reaction should be determined, and, if positive, a course of Quinostab followed by small doses of neoarsphenamine (up to 0.45 G.) is given (see p. 248). It is not now considered necessary to give the high protein diet recommended by Epstein. For an adult a diet may be given containing protein 100 to 130 G., carbohydrate 250 G. and fat 80 G. A child requires protein 2.3 G. per kg. bodyweight and enough fat and carbohydrate to yield a high calorie diet. Fluid should be restricted to 40 to 45 oz. No salt must be used in cooking or added to the food.

A preliminary venesection followed by a blood transfusion assists in restoring the blood proteins to their normal value.

Thyroideum may also be given, beginning with gr. 1 t.d.s. and working up to large doses such as gr. 15 to 40 daily, as long as the blood cholesterol is raised. This is especially indicated in cases with a low metabolic rate.

In obstinate cases concentrated human blood serum or plasma may be used as a diuretic. The dried powder obtained from the serum or plasma is put up in a tube with a rubber cap, each tube being marked with the number of mls of original serum from which it was obtained. Sterile water is injected with a syringe and needle through the rubber cap, one quarter the volume of the original serum being injected. Twenty to 60 mls are given slowly intravenously at a rate of not more than 5 mls a minute. The treatment is repeated once or twice at intervals of 2 or 3 days. The best results are obtained when the treatment is given within 6 weeks of the onset of the œdema. Salyrgan (mersalylum B.P.) may be administered cautiously together with ammon. chlorid. (see p. 228) if there is no nitrogen retention. Other diuretics which may be tried include urea 15 G. (gr. 225) in 5 oz. of water flavoured with syrup of orange twice daily for 7 to 10 days. The legs, abdomen and thorax may also be drained if necessary. If these methods fail a trial should be given to those described under the heading "Chronic Diffuse Glomerulo-nephritis" (see p. 455).

### Amyloid Nephrosis

**Etiology.** Amyloid nephrosis may occur in connection with chronic pulmonary tuberculosis, syphilis, or chronic suppuration.

**Pathology.** The kidney is enlarged, smooth and pale. The capsule strips readily. The glomeruli show on section as dark brown spots, when stained with iodine. Later, atrophy and fibrosis result in a small firm contracted kidney. Microscopically it is seen that amyloid is deposited around the capillaries and in the walls of the arterioles and

venules The glomerular vessels may be the only ones affected Fatty and atrophic changes occur in the cells of the first convoluted tubules

**Clinical Findings** Oedema develops, and the urine often contains a considerable amount of albumin There may be polyuria The blood changes resemble those found in lipoid nephrosis The total protein may be as low as 4%, due chiefly to loss of albumin The blood cholesterol is raised, 300 to 600 mg per 100 c c The nitrogen figures are normal, except in cases of the amyloid contracted kidney in which there is azotæmia

**Treatment** No special measures are indicated for the nephrosis, the underlying cause being the essential factor for treatment. A sufficiency of protein should be given in the diet to compensate for the protein lost in the urine

### Nephrosclerosis

#### (Chronic Interstitial Nephritis)

Under this heading are included 1 Benign renal arteriosclerosis 2 Malignant renal arteriosclerosis 3 Senile renal arteriosclerosis

### Benign Renal Arteriosclerosis

#### (Primary Ischæmic Nephritis)

**Etiology** Benign renal arteriosclerosis is an accompaniment of essential hypertension (see p 66)

**Pathology** The changes in the renal arterioles are secondary to hypertension The hypertension possibly is secondary to a long standing hypertonus of arteriole muscle fibres widely distributed throughout the body : e a functional vasoconstriction There is first a hyaline degeneration beneath the endothelium of the arterioles which narrows their lumen later there is atrophy of muscle fibres and replacement fibrosis These changes correspond with Gull and Sutton's arteriocapillary fibrosis (see p 62) The kidneys In the early stage the arteriosclerosis is the only change Later the kidney is small and granular due to fibrous tissue formation and cysts may be present on the surface The capsule is adherent The kidney is tough on section and reddish (red granular kidney) The cortex is diminished Areas of atrophied kidney substance are present, in which both glomeruli and tubules are affected

**Clinical Findings** The patient is usually an adult male over the age of 50 who complains of weakness dyspnoea, headache palpitations or precordial pain He may have had a temporary weakness of one or more limbs or a transitory aphasia These symptoms are due to hypertension and hypertensive heart disease There may be frequency of micturition at night

**On Examination** The heart may be hypertrophied the arteries are thickened the blood pressure raised the systolic pressure being about 200 mm Hg, but the diastolic is usually not over 120 mm Hg Ophthalmoscopic examination may show the changes of retinal arteriosclerosis, retinal hæmorrhages with tortuous arteries passing over

constricted veins. The urine: The volume may be slightly increased, with a relative increase in the amount passed by night. Sp. Gr. 1,005 to 1,012. A trace of albumin. Deposit, a few epithelial and blood cells and an occasional hyaline and granular cast. The blood: Urea and non-protein nitrogen, cholesterol, and protein are normal.

The urea concentration test is normal, but the water elimination test may indicate deficiency.

**Differential Diagnosis.** The benign type is differentiated from malignant renal arteriolosclerosis by the lower diastolic blood pressure, the milder course of the disease and the normal renal function tests.

**Course and Complications.** These are those of cardiac hypertrophy and arteriosclerosis (see p. 223).

**Prognosis.** This is dependent upon the state of the heart and blood-vessels, and not on the renal function.

**Treatment.** This is directed to the cardio-vascular system (see p. 267). No particular treatment is necessary for the albuminuria.

### Malignant Renal Arteriolosclerosis

**Pathology.** The malignancy of this condition is in some cases due to very extensive renal changes similar to those described as occurring in the benign type, but in others it is due to necrosis and endarteritis of the renal arterioles. The kidneys: They may be normal in size, slightly larger than normal, or somewhat contracted. Granulations are usually slight. The colour may be dappled by red and yellow areas. In addition to the changes in the arterioles and degeneration of the glomeruli and tubules, hæmorrhages may be seen.

**Clinical Findings.** The patient is usually an adult between the ages of 30 and 40, and more often a male. The condition may occur in children. The onset is often insidious and resembles that described above for the benign type. In some cases the onset is sudden with a cerebral hæmorrhage or uræmic attacks.

**On Examination:** The heart is hypertrophied and the arteries thickened. The blood pressure is high, the systolic being usually over 200 mm. Hg., and the diastolic between 120 and 160 mm. Hg. Ophthalmoscopic examination: Hypertensive neuro-retinopathy (albuminuric retinitis) is usually present. The disc is swollen, red, and its edges blurred. The retina shows fluffy white spots ("cotton wool" areas) due to exudate and fatty changes. Hæmorrhages are present. The arteries are narrowed ("silver-wire" appearance) and the veins are dilated. The urine: The volume may be as much as 80 or 100 oz., with an increase in the nocturnal output. In the later stages the excretion falls and a fixed amount tends to be passed every hour by day and night. Sp. Gr. 1,002 to 1,008. Protein, a trace to 0.5%. Deposit, occasional renal epithelial and red cells, and hyaline and granular casts. The blood: Urea and non-protein nitrogen are increased to 100 mg. per 100 c.c. or more. Cholesterol normal. Creatinin may be raised to 2.5 mg. per 100 c.c. Calcium may be low, 6 mg. per 100 c.c. or less. The urea concentration and water elimination tests show low figures as the disease progresses.

**Differential Diagnosis.** The high diastolic pressure, the progressive course of the disease, the retinal changes, and the evidence of failure of renal function differentiate the malignant from the benign renal sclerosis

**Course and Complications** The course is slowly progressive, a cerebral hæmorrhage, uræmia, heart failure, and secondary infections such as bronchopneumonia are liable to occur. Terminal pericarditis and pleurisy are probably toxic in origin. Acute œdema of the lungs is rare, but attacks of nocturnal dyspnœa (renal asthma) with Cheyne-Stokes breathing may occur. Exacerbations of an acute nephritic type may also be noted, with œdema, increase of the albumin, and the presence of more red cells in the urine

**Prognosis** This is always very grave, although the patient may live for several years. Unfavourable signs are a blood urea figure of over 200 mg per 100 c c and the presence of "albuminuric retinitis". In 90% of cases death occurs within two years of the diagnosis of the latter

**Treatment** The general regime consists in regular hours, moderation in work, exercise diet, alcohol and smoking. The two latter are best avoided completely. The most suitable climate for the winter is that of Egypt, but if it is not possible to go abroad all chills should be avoided. Diet. The protein allowed is determined to a certain degree by the blood nitrogen figures. The basal requirement is about 0.5 G of protein for every 1 lb of body weight or 70 G for a man of 10 stones. With a blood urea of about 80 mg per 100 c c the patient can be put on the moderate protein diet (see p 155)

With a blood nitrogen of over 100 mg per 100 c c, a low protein diet containing about 35 to 40 G of protein and a caloric value of 1,700 can be given for periods of 2 or 3 weeks alternating with the moderate protein diet

**Low-protein Diet** Breakfast Oatmeal (uncooked), 1 oz, bread 2 oz, butter,  $\frac{1}{2}$  oz, milk 4 oz, tea  $\frac{1}{2}$  oz. Lunch Green vegetables, 2 oz, potato 4 oz, bread 2 oz, butter,  $\frac{1}{2}$  oz, stewed fruit, 2 oz, cream,  $\frac{1}{2}$  oz, jam,  $\frac{1}{2}$  oz, rice (uncooked)  $\frac{1}{2}$  oz. Tea: Bread, 1 oz, butter,  $\frac{1}{2}$  oz, tea  $\frac{1}{2}$  oz, jam  $\frac{1}{2}$  oz, milk, 2 oz. Dinner Vegetable soup, white fish or lean meat or chicken (cooked), 2 oz, potato, 4 oz, fruit 4 oz, bread, 1 oz. Sugar for sweetening during the day, 2 oz

While the patient is on the low diet he should be in bed. The bowels should be kept acting freely with the help of salts (mag sulph or sod sulph gr 60 to 120) in the morning and a weekly dose of calomel gr 3 at night. For insomnia chloral hydrate gr 5 and sod brom gr 10 may be given at night. For headache with high blood pressure tabella glyceryl trinitrat gr 1/130 may be given 3 times a day, but it is not wise to endeavour to lower the blood pressure considerably. For dyspnœa due to acidosis alkalis such as sod bicarb gr 20 may be given six hourly, and if there is heart failure with œdema the digitalis may be given in doses of m 5 to 10 t d s. The treatment of uræmia is considered later (see p 167).

### Senile Renal Arteriosclerosis

This is associated with senile arteriosclerosis. The kidneys are scarred by infarcts and are contracted.

**Clinical Findings.** The arteries are thickened, but the blood pressure is not raised. The urine often contains a trace of albumin, but the renal function tests are normal. No special treatment is required for the kidney lesions.

### Mixed Types of Chronic Nephritis

Various mixed types of chronic nephritis are met with. Thus a patient may have chronic nephritis with œdema, and the blood nitrogen figures are found to be raised, as well as the cholesterol. Another patient with chronic nephritis without œdema may have a high blood cholesterol and approximately normal blood nitrogen. Further, nephritis may complicate nephrosis, as shown by the presence of blood in the urine, and hypertension. A high protein diet should not be given when the blood nitrogen is raised.

## URÆMIA

**Definition.** Toxæmia due to, or associated with failure of renal function.

**Etiology.** The cause is uncertain, and it probably differs in the various types of uræmia. A distinction may be drawn between prerenal, renal, and postrenal causes of uræmia.

**Prerenal causes.** The primary changes occur in tissue metabolism, in the chemical composition of the blood, and in the circulation. There is probably secondary renal insufficiency, although no structural changes may be demonstrable in the kidneys. The causes may be listed as follows: Vomiting due to pyloric and intestinal obstruction, and hyperemesis gravidarum. Diarrhœa, especially infantile and that due to cholera. Hepatic disturbances, as after operations on the gall-bladder and bile-duets, and cholemia associated with acute hepatitis and cirrhosis. Diabetes mellitus with acidosis, especially in untreated cases. Addison's disease during a crisis. Severe hæmorrhage, particularly hæmatemesis. Post-operative and traumatic shock. Severe burns. Peripheral circulatory collapse, as in diphtheria and pneumonia. Coronary thrombosis associated with shock. Overdosage with alkalis in the treatment of peptic ulcer. A mismatched blood transfusion and the crush syndrome.

**Renal causes.** These include inflammatory, degenerative, and developmental changes in the kidneys, such as acute and chronic nephritis, bilateral renal suppuration, necrotising nephrosis, and congenital cystic disease.

**Postrenal causes.** There is obstruction to the outflow of urine. This may be due to mechanical causes such as calculi blocking both ureters, a calculus obstructing the sole-functioning kidney, removal of the only existing or functioning kidney, accidental ligature of both ureters in a pelvic operation, pressure of a pelvic carcinoma on both



ureters, or a vesical growth obstructing the orifices of both ureters. Incomplete obstruction may result from an enlarged prostate, a pelvic tumour, or a bilateral hydro or pyonephrosis. Reflex inhibition of urinary secretion may result from a drainage tube placed in the bladder after prostatectomy.

**Pathogenesis** Various theories have been propounded to account for the toxic and cerebral symptoms of uræmia. These include: 1. *Azotæmia* (accumulation of urea and other non-protein nitrogenous substances in the blood) Urea, if given to man in large doses, is toxic and produces symptoms resembling uræmia. In many cases of uræmia these substances are much raised in the blood. On the other hand, high figures may be obtained without there being any uræmic symptoms, and uræmia may ensue when the figure is not higher than 100 mg. per 100 c c. Azotæmia is one factor in the production of uræmia. The precursors of urea, ammonium carbamate and ammonium carbonate, are usually considered to be non toxic. Ammonia is not present in the blood in excess in uræmia.

2 *Cerebral Oedema* Traube introduced the view in 1871 that a mechanical pressure on the brain due to oedema would account for certain uræmic manifestations. After being discredited, this theory has again been revived and it is now believed by many authorities that local oedema of the brain is the cause of focal nervous symptoms such as convulsions, and of coma and amaurosis in certain cases of acute uræmia (hypertensive encephalopathy).

3 *Cerebral Vascular Spasm* This can cause convulsions, localised paralyses, temporary amaurosis, or coma. It appears probable that the cerebral vessels have vasomotor nerves. The vessels may contract as the result of the local stimulus of the increased intravascular tension. This would account for cases of hypertensive encephalopathy, in which post-mortem the brain is dry.

4 *Deficiency of Blood Calcium*. This probably accounts for the muscular twitchings in chronic uræmia.

5 *An Unknown Toxic Substance* Trimethylamine is present in the blood in uræmia and on injection into certain animals produces uræmic like convulsions. Other toxic substances may also be present, such as intestinal putrefactive bodies, especially phenols.

6 *Acidosis* This is usually present, due to retention of acid metabolites, and probably accounts for respiratory symptoms such as renal asthma, and other types of dyspnoea.

7. *Alkalosis* This results in azotæmia.

8. *Severe Anæmia and Renal Anoxia*. This causes an impairment of renal function.

9. *Lack of an Internal Secretion of the Kidney*. Such a secretion has not been isolated.

10 *The Presence of Nephrolysins or a Toxic Substance produced in the Kidney*. A pressor substance, renin, may be produced in an ischæmic kidney, resulting in high blood pressure.

11 *Loss of Water and of Mineral Salts*. This may be a factor in some cases.

The following changes may be expected in the blood when there is failure of renal function, not all being present in each case: *An increase* of urea, non-protein nitrogen, uric acid, creatinin, indican, phosphate, and sulphate. *A decrease* of calcium, chloride and sodium (if there is vomiting). The ammonia and amino-acid contents are not usually affected, and frequently there is acidosis especially in chronic renal disease, diabetes mellitus and Addison's disease. Alkalosis is generally associated with vomiting or overdosage of alkalis. Uræmia never occurs unless there is azotæmia, but the converse is not true.

*Prerenal Uræmia.* Increased protein destruction may be a factor in the production of azotæmia, but as the body in health can eliminate large quantities of urea, there must be in addition some impairment of renal function. In many of the conditions, such as hæmorrhage and shock, there is a fall in blood pressure which reduces the glomerular filtrate, and also renal anoxia which causes damage to the renal epithelium. In Addison's disease there is further an alteration in the electrolytic balance of the blood and a rise in the hydrogen ion concentration. Excessive vomiting may produce azotæmia before alkalosis occurs, owing probably to loss of chlorides. In alkalosis due to excessive intake of alkalis, there is presumably some renal impairment. In a mismatched blood transfusion and in the crush syndrome renal failure results from blockage of tubules with epithelial and blood pigment debris, and damage to tubular epithelium.

In some of these conditions the urine may appear normal, but simultaneous observations on the specific gravity and volume of urine will indicate renal impairment.

*Renal and Postrenal Uræmia.* It is usually said that it is necessary for three-quarters of the renal parenchyma to be out of action before azotæmia occurs; but if there is a feeble circulation due to cardiac failure or peripheral circulatory failure associated with severe infections, renal failure may ensue with lesser degrees of renal damage. In both these types of uræmia the main cause appears to be retention in the blood of normal urinary constituents.

It is probable that the acute convulsive and comatose type of uræmia (false uræmia) met with in acute glomerulo-tubular nephritis, is due to high blood pressure, arterial spasm, and cerebral œdema. According to this view these phenomena are not uræmic, but constitute hypertensive encephalopathy.

The three clinical types of uræmia will now be considered.

### Acute Uræmia

Two types may be described, *false* associated with acute glomerulo-nephritis, and *true* met with in acute hæmoglobinuric nephrosis.

#### Acute Convulsive, Epileptiform or Eclamptic Uræmia

(*False Uræmia. Hypertensive Encephalopathy*)

**Clinical Findings.** The patient is usually suffering from acute glomerulo-nephritis, but acute symptoms may occur in association with

## Chronic Uræmia

## (Uræmia)

**Clinical Findings.** It is usually known that the patient is suffering from chronic nephritis. The onset of uræmia is generally insidious, and the symptoms may be grouped under the headings of the gastro-intestinal, nervous, and respiratory systems. *Gastro-intestinal symptoms:* There is thirst, an unpleasant taste, which may be bitter or ammoniacal due to excess of urea and ammonia in the saliva, and loss of appetite. There may be nausea, vomiting, diarrhoea associated with uræmic ulceration of the colon, or constipation. *Nervous symptoms:* The patient may complain of general weakness, inability to concentrate, and insomnia. Persistent, dull headache is often an early symptom, in addition to which muscular twitchings may occur. Later, there may be hiccough, convulsions, amaurosis, and finally coma. *Respiratory symptoms:* Various types of dyspnoea are described, such as nocturnal attacks (renal asthma), Cheyne-Stokes breathing, or air hunger (Kussmaul respiration). The "hissing" breathing is not often heard. *Cardiac symptoms:* Cardiac failure and terminal pericarditis may occur. The skin is often dry, yellowish, and pruritus is troublesome. A skin "frost" (urea crystals) is rarely seen. The temperature is usually subnormal and the patient is emaciated and dehydrated. The blood and cerebro-spinal fluid show a rise in urea and non-protein nitrogen.

**Differential Diagnosis.** If the non-protein nitrogen of the blood is low in patients presenting the signs of chronic nephritis, it can usually be assumed that chronic uræmia is not present. Alkalosis can be confirmed by determining the alkali reserve of the blood.

**Prognosis.** This depends upon the cause, which sometimes can be removed, as in the case of an enlarged prostate. Some of the conditions, such as alkalosis due to overdosage with alkalis, respond rapidly to treatment (see p. 38). In others, such as malignant renal arteriosclerosis, the outlook is hopeless.

**Treatment.** This must be directed primarily to the causative condition and secondarily to the relief of symptoms. If the blood nitrogen figures are high, a preliminary venesection followed by a saline intravenous infusion usually affords temporary relief. The patient should also be placed on a low protein diet (see p. 462) for 3 or 4 weeks, this can then be alternated with a medium protein diet (see p. 455). The bowels should be kept open with saline aperients, but intractable diarrhoea ensues in some cases. Various remedies may be tried for the nausea and vomiting, such as liq. iodi mut. m. 1 to 2 in an ounce of water, every hour, or a mixture containing Ceri oxalat. (B.P.C.) gr. 5, bism. carb. gr. 10, liq. adrenalin. hydrochlor. m. 10, acid. hydrocyan. dil. m. 3, sp. chlorof. m. 5, and aq. menth. pip. dest. ad fl. oz. 1. Fl. oz. 1 t.d.s. Washing out the stomach with normal saline is also helpful in some cases. Rectal injections of 4 to 8 fl. oz. of normal saline containing 5% dextrose may be given every 4 hours, with small drinks of water by mouth. Insomnia and headache are best treated by chloral hydrat.

gr 10 and sod brom gr 20 tds, or tabella glyceryl trinitrat. gr 1/130 may be given tds to lower blood pressure. If the blood calcium is low, muscular twitchings can be stopped in some cases by the intravenous injection of 5 mls of calcium gluconate (B.P. Add.) For hiccough an injection of morphin sulph gr  $\frac{1}{2}$  may be given. There is no cure for the terminal coma, but venesection may be tried.

### Latent Uræmia

#### (*Urinæmia*)

This occurs when there is complete failure of renal function, and is due to postrenal mechanical causes (see p 463). The symptoms are entirely due to retention of urinary substances, there is no element of dehydration or of hypertensive encephalopathy. Beyond anuria there may be no symptoms noted for the first 4 or 5 days. The patient then becomes a little drowsy, the temperature falls and the pupils become smaller. The blood pressure does not rise. The blood urea and non-protein nitrogen figures are increased to over 200 mg per 100 c.c., and the alkali reserve falls to about 30 c.c.  $\text{CO}_2$  per cent. Vomiting and muscular twitchings may usher in the final stage of coma. Death usually occurs in from 10 to 14 days. Unless the obstruction such as a calculus, can be removed, no treatment is available.

## BACTERIAL INFECTIONS OF THE RENAL TRACT

### Bacilluria

**Definition.** The presence of bacteria in the urine, not accompanied by local symptoms.

**Etiology.** The organisms may gain access to the urine from the blood, being excreted by the kidneys, from the intestines through the lymphatics in connection with the kidneys, or by an ascending infection of the urethra. Excretion of organisms by the kidneys implies a certain degree of renal damage. Such organisms as the *Bacterium commune* (B coli) the *Proteus vulgaris* (B proteus) the *Bacterium typhosum* (B typhosus) the *Bacterium paratyphosum* A, B or C (B paratyphosus A, B or C), the *streptococcus* the *staphylococcus*, the *Neisseria gonorrhoea* (gonococcus), or the *Diplococcus pneumoniae* (pneumococcus) may be found. Chronic constipation is not a predisposing cause. In women habitual looseness of the bowels may be of etiological significance.

**Clinical Findings.** The history is of value. Thus the bacilluria may be a sequel of an acute illness, such as typhoid fever, or occur during an acute illness, such as pneumonia, infective endocarditis, or gonorrhoea. It may also be noted in cases whose chief symptom is debility, as in certain B coli infections, or there may have been no symptoms attributable to the bacilluria.

**Treatment.** This depends upon the associated conditions, for which the special sections should be consulted.

calculus, renal tuberculosis, obstruction of the ureters due to a calculus or abdominal tumour, cystitis, or urethral obstruction

*Varieties* • Pyelitis may be acute or chronic.

### Acute Pyelitis

**Clinical Findings** The patient, who is suddenly taken ill, complains of malaise due to the fever and toxæmia. There may be no other symptoms, or she may complain of aching or tenderness in the loin with frequency of micturition. There may also be rigors.

**On Examination**, The temperature is found to be raised, and there is usually abdominal tenderness over the affected kidney. The urine shows the changes described above.

**Differential Diagnosis.** Acute pyelitis is liable to be mistaken for appendicitis, salpingitis, or, if occurring after childbirth, for puerperal septicæmia. The diagnosis is established by the examination of the urine.

**Course and Complications** The course depends largely on the treatment given. Relapses are liable to occur or chronic pyelitis may supervene.

**Prognosis** This is on the whole favourable.

**Treatment** The patient should be put to bed and a purge given, such as castor oil  $\text{fl oz } \frac{1}{2}$  followed by mag sulph gr 120 next morning. Subsequently the bowels should be kept open daily with Cascara Laxant (m 30 to 60) or with an enema.

**The Diet** Too much milk must not be given, but the patient should take plenty of fluid (6 to 8 pints daily) such as water, fruit juices and barley water. Hot applications such as Antiphlogistine (cataplasma kaolin B.P.) should be placed on the loin if it is painful. The urine should be made alkaline by giving a mixture of Sod bicarb gr 30, sod cit gr 30, pot acetat gr 30, syr aurant m 30, aquam ad  $\text{fl oz } 1$  Fl oz 1 four hourly. As soon as the urine is alkaline the temperature usually falls and the mixture can be given six hourly and then 3 times a day as required. Hexamine gr 10 in a glass of water should now be given t.i.d. i.e., and after food a mixture of Acid sod phosph gr 30, tnc hyoseyam m 30, aq chlorof ad  $\text{fl oz } 1$  Fl oz 1 t.i.d. The urine must be kept acid while hexamine is being given, so that it can exert its antiseptic action. The acid and alkaline treatment should be alternated every 3 days. In a favourable case, diagnosed early, the organisms disappear from the urine in a week or so. A vaccine should be prepared from the urine for subsequent use if required.

An alternative method of treatment consists in the administration of mandelic acid. This may be used even in acute cases with high temperatures. The urine must be kept acid at or about a pH of 5.4. The total fluid intake during each 24 hours must be limited to two pints. The treatment should be continued for one or two weeks. The urine should be examined every third day for albumin, red cells and casts. A satisfactory and simple way of giving the treatment is as follows. Two prescriptions are made up. *Mixture A* Mandelic acid gr. 40 (3 G), ammon bicarb gr 23 (1.48 G), ext glycyrrhiz liq m 5, sp

chlorof. m. 7, sacchar. sol. gr.  $\frac{1}{2}$ , syr. aurant. m. 90, aquam ad fl. oz.  $\frac{1}{2}$ .  
*Mixture B.* Acid. sod. phosph. gr. 15, aq. chlorof. ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  of mixtures A and B are mixed and taken four times a day after food. The reaction of the urine is tested first thing in the morning, at noon, and in the evening, by adding two drops of methyl red indicator to 2 c.c. of urine. One of three colours may be obtained, yellow (alkaline), delicate pink (correct acidity), or deep red (too acid). If the colour is yellow more of mixture B should be given, if it is red mixture B should be omitted. If red cells or casts appear in the urine, the treatment should be continued with caution, daily examinations being made to see if they increase in number. Usually they disappear when the treatment is discontinued and no permanent damage is done to the kidneys. If the urine is not sterile at the end of seven to ten days, the treatment should be continued for a fortnight. If organisms are still present the treatment has failed. The urine should be examined again a month after the termination of a successful course of treatment. If organisms have reappeared a second course should be given. Alternatively calcium mandelate may be given in the form of Mandecal, one level dessertspoonful contains 3 G. of mandelic acid. This dose is administered in water 4 times a day. It is seldom necessary to give additional ammonium chloride to lower the pH of the urine to 5. Sulphanilamide or Sulphapyridine may be used for *B. coli* and *B. proteus* urinary infections, and Sulphathiazole for those due to the streptococcus faecalis and staphylococcus aureus. Two 0.5 G. tablets are given t.i.d. for 5 to 7 days. The patient must remain in bed, and no restriction of fluid intake is required.

### Chronic Pyelitis

#### (Relapsing or Quiescent Pyelitis)

**Clinical Findings.** Often there are no local symptoms, but the patient may complain of frequency of micturition and irregular attacks of fever with malaise.

**Differential Diagnosis.** This is established by ureteric catheterisation. An X-ray examination should be made to exclude a renal calculus, and the urine also examined for tubercle bacilli.

**Treatment.** A course of mandelic acid or of a sulphonamide drug should be given as described above. If the infection still persists the question of lavage of the renal pelvis with an antiseptic solution such as 1 in 15,000 silver nitrate should be considered.

### Cystitis

**Definition.** Inflammation of the bladder.

**Etiology.** Cystitis is due to infection with organisms such as the *Bacterium commune* (*B. coli*), the staphylococcus, the streptococcus, the *Neisseria gonorrhoea* (gonococcus), the *Mycobacterium tuberculosis* (*B. tuberculosis*), and the *Bacterium typhosum* (*B. typhosus*). It may be associated with vesical bilharziasis, a calculus, adhesions between the bladder and colon or a diverticulum, pyelitis, enlargement of the

prostate, an urethral stricture, pelvic tumour, retroverted gravid uterus, spinal cord lesions, etc. *Predisposing causes* Chill, constipation and over indulgence in alcohol, and incomplete emptying of the bladder at micturition

**Clinical Findings** *Acute Cystitis* The patient is suddenly taken ill with severe pain in the hypogastrium or perineum, and he has to pass urine every 3 or 4 minutes, only about a teaspoonful being evacuated at a time. There is usually no fever and no constitutional disturbance. *Chronic Cystitis* The symptoms are less marked, but there is often an aching pain over the pubes or in the perineum and frequency of micturition. The urine. An acid reaction implies infection with the B coli, B tuberculosis or the gonococcus. The urine is alkaline with the other infections and in any mixed infection. It is cloudy, contains a trace of albumin, and blood may be present. The deposit shows vesical epithelial cells and often some pus cells. In children a staphylococcal infection is not uncommon. The parent notices that the urine is thick or contains slimy or stringy matter, which may only be present in the morning specimen.

**Treatment.** *Acute Cystitis* The patient should be kept in bed and hot applications placed over the hypogastrium. The diet and regulation of the bowels are as for the treatment of pyelitis (see p 470). If the urine is acid, an alkaline mixture should be given with the hyoseyami m. 30 six hourly. If it is alkaline, acid. sod phosph. should be administered, such as Acid sod phosph gr 30 to 60, the hyoseyami m. 30, sp chlorof m 7, aquam ad fl oz 1 Fl oz 1 in water six hourly. When the urine is acid, hexamine should be given before meals in water, as for pyelitis (see p 470). In many cases mandelic acid, administered as described on p 470, is the most successful form of treatment. Tab acriflavin (B P C) gr  $\frac{1}{2}$  may be given daily, being an antiseptic which acts in an alkaline medium.

*Chronic Cystitis* In addition to the above measures, the bladder may be irrigated with a 1/12,000 solution of silver nitrate, and an autogenous vaccine given weekly, beginning with small doses such as 250 000 organisms.

### Pyelonephritis

**Definition** Inflammation and suppuration involving the renal pelvis and kidney substance.

**Etiology** The infections may be ascending and secondary to pyelitis, or blood borne in pyæmia, or due to lymphatic spread from an appendix or retroperitoneal abscess.

**Pathology** Multiple abscesses may be seen on the surface and in the substance of the kidney, with inflammation of the pelvis.

**Clinical Findings** The patient has usually the symptoms of pyelitis (see p 470) with periodical attacks of fever and rigors. Acute cases occur which may prove fatal in a week or so. The urine contains organisms, albumin, pus and blood.

**Treatment.** This is as for pyelitis. Surgical drainage of the kidney is required in some cases.

### Pyonephrosis

**Definition.** Distension of the renal pelvis with pus.

**Etiology.** Pyonephrosis results from infection of the renal pelvis, together with obstruction to the outflow of urine. The latter may be due to a stricture, enlarged prostate, diverticulum of the bladder, vesical calculi, growth, etc.

**Pathology.** One or both kidneys may be affected, depending largely on the site of the obstruction.

**Clinical Findings.** The patient complains of malaise, and usually of aching or pain in the loins.

**On Examination:** A renal tumour may be felt on one or both sides. The temperature is usually raised. The urine contains pus, albumin and organisms. If both kidneys are affected there is likely to be an increase in the blood urea and non-protein nitrogen.

**Treatment.** This is surgical.

### • Perinephric Abscess

**Definition.** Suppuration in the tissues around the kidney, usually in the pararenal fat.

**Etiology.** The infection may be blood-borne, the focus being in a cutaneous boil or carbuncle, or in the tonsils. Lymphatic spread may occur from an infected kidney, gall-bladder, appendix, or from spinal caries.

**Clinical Findings.** The patient may complain of no symptoms except malaise due to fever, and at times joint pains. In other cases an aching or a sharp pain may occur in the loin.

**On Examination:** The temperature is found to be raised and it runs an irregular intermittent course. Tenderness is elicited in the loin, and later a definite bulging may be seen, or a swelling is felt on abdominal palpation over the kidney. The patient may lie with the hip joint flexed on the affected side, if the abscess is tracking downwards. If, on the other hand, it is situated above the kidney, the diaphragm on the affected side may be raised. This results in some dulness and weak air entry over the base of the corresponding lung, but tactile fremitus is present. An X-ray examination will also reveal the position of the diaphragm. The blood: A leucocytosis of about 12,000 to 20,000 per c.mm. may be present. The urine contains a trace of albumin, some pus cells and organisms (usually staphylococci) if the kidney is also affected. The abscess may eventually point in Petit's triangle or rupture intraperitoneally.

**Differential Diagnosis.** The history of a crop of boils or carbuncles is suggestive, but if there are no localising symptoms, other causes of obscure pyrexia have to be considered, and the diagnosis is not usually made until a swelling appears. In the early stages the persistent fever, joint pains and constipation are suggestive of infective endocarditis or of a *Brucella abortus* infection (see p. 559). The agglutination test on the blood will exclude the latter.

**Treatment.** This is surgical, the abscess being drained.



## Genito-urinary Tuberculosis

Tuberculosis may affect the kidneys, ureters, bladder, or the genital organs such as the *epididymis testis*, seminal vesicles, prostate, Fallopian tubes and, rarely, the uterus and ovaries

The majority of these conditions are surgical, and occur in adults below the age of 40, but children may be affected in cases of miliary tuberculosis

## Tuberculosis of the Kidney

**Pathology** In the majority of cases the infection is blood borne, ascending infection from the bladder being rare. The primary focus may be in the lungs, lymph glands or elsewhere, but the site is often unrecognisable clinically

**Clinical Findings** The patient may complain of aching in the loin, frequency of micturition and blood in the urine

**Differential Diagnosis** If the urine contains tubercle bacilli and pus cells, renal tuberculosis is almost certainly present. Cystoscopy and catheterisation of the ureters are necessary to establish the site of the lesion

**Treatment.** This consists either in administration of tuberculin, beginning with B.E. 1/500 000 mg and gradually increasing the dose at weekly intervals to 1/5,000 mg, for 6 to 9 months, or in the surgical removal of the kidney. One kidney should not be removed if both are infected. It is unwise to administer tuberculin, if there is co-existent pulmonary tuberculosis

## Tuberculous Cystitis

This is usually secondary to tuberculosis of the kidney, epididymis or prostate. The primary source of infection should be removed surgically, if a satisfactory response is not obtained with tuberculin injections

## Hydronephrosis

**Definition** Dilatation of the renal calyces and pelvis

**Etiology** Hydronephrosis may be congenital or acquired, and is due usually to a gradual or intermittent obstruction to the outflow of urine

**Congenital Hydronephrosis** The obstruction may be caused by an abnormal branch of the renal artery, by a disordered function of the pelvi-ureteral junction or by an imperforate urethra. It is usually bilateral

**Acquired Hydronephrosis** The causes may be grouped as follows  
 1 Renal. A calculus or growth in the pelvis  
 2 Ureteric. A disorder of the nervous control of the pelvi-ureteral junction. Kinking of the pelvi-ureteral junction by a movable kidney or renal tumour. A calculus or stricture.  
 Pressure due to a pelvic or abdominal tumour, or adhesions.  
 Injury during an operation.  
 3 Vesical. A calculus or tumour.  
 4 Urethral. A stricture, calculus, enlarged prostate or phimosis

**Pathology.** The kidney substance atrophies and only a thin covering

may remain. In internal hydronephrosis the calyces are dilated, but later, the pelvis distends and the kidney substance may be replaced by a sac as large as a cocoa-nut, the ureter also dilating to the level of the obstruction. In intermittent hydronephrosis, which is usually associated with a kinking of the ureter, the pelvis dilates and contracts as the obstruction appears and disappears. Bilateral hydronephrosis is due to obstruction in the urethra or bladder.

**Clinical Findings.** *Intermittent Hydronephrosis.* The patient is usually a woman, who complains of periodical attacks of pain in the loin, generally on the right side, and nausea and vomiting, often associated with fever. There is scanty micturition and relief comes with the passage of large quantities of urine. The pain may be referred to the healthy side by a reno-renal reflex. Kinking of the ureter may give rise to pain resembling renal colic, which is known as a Dietl's crisis (see p. 478).

**On Examination:** A renal tumour may be felt which disappears with the onset of the polyuria.

**Continuous Hydronephrosis.** The symptoms are less marked, there is some aching in the loin. If, however, the condition is bilateral and progressive, symptoms of chronic uræmia gradually ensue.

**On Examination:** A renal tumour may be felt. The blood: A rise in the nitrogen figures indicates that both kidneys are affected.

**Differential Diagnosis.** The hydronephrosis may be demonstrated in some cases by pyclography. If more than 20 mils of 15% sodium iodide can be injected into the renal pelvis, hydronephrosis is held to be present. Radiography after intravenous injection of Uroselectan B (iodoxylum B.P.Add.) may also demonstrate the lesion.

**Course and Complications.** The course must vary with the nature of the obstruction. Pyonephrosis may occur as a complication.

**Prognosis.** This also varies with the cause. Bilateral lesions prove fatal unless the obstruction is removed before irreparable and extensive renal damage has been done, death occurring from uræmia or suppuration.

**Treatment.** If the hydronephrosis is due to a movable kidney a renal support should be tried. If this does not afford relief an operation will probably be necessary. In the majority of cases the treatment is surgical.

### Urinary Calculus

**Definition.** Stone in the kidney, ureter or bladder.

**Etiology.** The cause of calculus formation is not known. Certain factors undoubtedly play an important part. These include infection, urinary stasis, a concentrated urine, and the presence of irreversible colloids, such as fibrin, which after precipitation will not go back into solution. Overdosage with alkalis, as in the treatment of peptic ulcer, may lead to the formation of phosphatic calculi. It is not known whether calculi are associated with a deficiency of vegetables in the diet, and it is uncertain why they are prone to occur in England in certain localities, as in Norfolk. In India, stone occurs amongst the poorer members of the community who live chiefly on cereals and

do not have sufficient milk This is possibly due to lack of vitamin A. They are not believed to be associated with chalky water Calculus formation usually occurs after the age of 20, males being most often affected, but urates may be deposited in the kidneys of infants, and vesical calculi found in the bladders of poorly nourished children Occasionally a large calculus is found in the kidney of a child under the age of 2 years One composed of sodium, magnesium and ammonium phosphate, and another of xanthine, have been removed

**Pathology** Calculi usually form around a colloid nucleus, such as fibrin, mucopus, cell debris and micro organisms Primary calculi are said to form in acid urine without any bacterial inflammation, whereas secondary calculi are deposited in alkaline urine infected with organisms Thus a phosphatic crust may be formed around an uric acid nucleus The following varieties are described *Uric acid* Brown, hard and irregular *Calcium oxalate* (mulberry calculus) Dark and irregular. When formed in the bladder they are round, they may be dendritic when occurring in the renal pelvis They are often mixed with calcium phosphate or uric acid *Ammonium urate* Hard and brownish *Triple-phosphate* Rather soft and smooth *Mixed or laminated calculi* They have an uric acid nucleus and a phosphatic coating *Cystine* Soft yellow green radially laminated *Xanthine* Reddish brown *Indigo* • Blue, will mark paper It is derived from indol and is extremely rare *Calcium carbonate* Smooth, hard and dark grey *Urostealth* Containing cholesterol

**Clinical Findings.** If the calculus remains in the kidney substance there may be no symptoms, or the patient complains of aching in the loin, and albumin and blood are found in the urine These symptoms may be related to jolting If the calculus obstructs the outflow of urine there is severe pain owing to stretching of the renal capsule This may occur with a stone in the ureter, and the pain is due to this distension rather than to the passage of the stone along the ureter The pain ceases either when the kidney does not secrete more urine, or when the obstruction is relieved The pain is felt in the flank and may be referred to the sound side, it may also radiate to the groin and inner side of the thigh There is usually a sense of nausea, and vomiting may occur Renal colic is an agonising pain, which is probably caused by muscular spasm of the renal pelvis and ureter The pain occurs in paroxysms and radiates along the course of the ureter to the groin, hypogastrium and testicle, and the testicle may be drawn up The patient is pale or flushed, sweats, rolls about in agony and may vomit There is a frequent desire to micturate, and the urine is scanty and may contain blood The attack lasts usually from a few minutes to 2 hours, but it may be considerably longer After the attack there is aching and tenderness in the loin

**On Examination** During the attack there is abdominal rigidity on the affected side The chief symptoms of a vesical calculus are attacks of pain felt at the end of the penis after micturition, radiating to the perineum and inner side of the thigh. There is also frequency of micturition and the urine shows evidence of cystitis (see p 472)

**Differential Diagnosis.** Renal colic must be differentiated from other varieties of abdominal colic, such as biliary, intestinal, appendicular or pancreatic colic, or from a Dietl's crisis associated with a movable kidney and kinking of the ureter, or rarely from a tabetic crisis. The X-rays will show renal calculi, provided they contain a sufficiency of calcium. Other shadows such as those caused by calcified glands and phleboliths must be excluded. Pyclography may thus be required in order to localise the shadow accurately.

**Course and Complications.** Several attacks of renal colic are not infrequent. The chief complications are due to : 1. *Infection*, resulting in pyelitis, pyelonephritis, pyonephrosis and cystitis. 2. *Obstruction*, producing hydronephrosis, pyonephrosis, renal atrophy and anuria if both kidneys are put out of action. 3. *Ulceration*, with extravasation of urine through the ureter. A stricture may subsequently form. 4. *Malignant disease*, affecting the kidney.

**Prognosis.** In some cases only one attack of renal colic occurs, but the prognosis is always serious, as the calculi tend to recur even after operative removal. The stone may be passed without an operation. If both kidneys are affected, or if there is evidence of failure of renal function, the outlook is very grave.

**Treatment. Prophylactic.** Plenty of fluids should be drunk in hot climates. Pyelitis should be adequately treated.

**Curative. During the attack :** Local heat applied to the loin or a hot bath may relieve the pain of a mild attack. For a severe attack an injection should be given of morphin. sulph. gr.  $\frac{1}{3}$  and atropin. sulph. gr.  $\frac{1}{100}$ . If the pain is not relieved inhalation of chloroform may be required.

**After the attack :** A mixture containing tnc. belladon. m. 15 and pot. citras gr. 30 should be given every 6 hours until the pupils dilate, to aid the passage of the calculus along the ureter. If the stone is not passed, the opinion of a surgeon should be obtained as to the advisability of operative removal. In any case the patient should be kept in bed until the hæmaturia has ceased, and an alkaline mixture given, if the urine is acid, containing Pot. cit. gr. 30, pot. bicarb. gr. 20, sod. bicarb. gr. 20, sp. chlorof. m. 7, aquam ad fl. oz. 1. Fl. oz. 1 t.d.s. **Subsequent treatment :** If oxalate or urate crystals are present in the urine, a dose of the alkaline mixture should be given at night, sufficient to render the morning specimen of urine alkaline. The diet should be rich in vegetables, and if urates are passed meat should only be taken in small quantities, and substances rich in nucleo-protein such as sweetbreads, kidneys and liver should be avoided. The diet recommended for oxaluria is described on p. 442. In all cases, at least 4 to 5 pints of bland fluids should be taken in the 24 hours.

### Movable Kidney

#### (Floating Kidney. Nephroptosis)

**Definition.** Undue mobility of the kidney.

**Etiology.** Normally the kidneys move with respiration 1 to  $1\frac{1}{2}$  inches. They are maintained in position by : 1. The perirenal fascia, which is attached to the diaphragm and encloses the kidney, the

suprarenal, and the perirenal fat 2 The renal pedicle, comprising the renal artery, vein and the ureter 3 The intra abdominal tension. (This however is normally negative) 4 The shape of the renal fossa, which is narrower at its lower end in the male than in the female

The factors which may cause undue mobility of the kidney therefore include 1 Weakness of the abdominal wall 2 Generalised visceroptosis and traction of a loaded colon 3 Enlargement of the kidney. 4 Scoliosis which renders the renal fossa more shallow 5 Emaciation 6 Obesity which may cause a mesenteric drag 7 The presence of a mesonephron 8 Trauma especially in athletes A movable kidney is more common in women owing to the greater prevalence of constipation and visceroptosis, the renal fossa is more shallow, and there is laxity of the abdominal wall after pregnancy Further, it is more likely to occur on the right side as the hepatic flexure causes a greater drag than the splenic flexure, and the renal fossa is more shallow on the right side The following degrees of mobility are recognised clinically  
*Palpable kidney* The lower pole can be felt on inspiration *Movable kidney* The upper pole can be felt on inspiration and the kidney held down with the finger above it *Floating kidney* The kidney can be pushed across the abdomen to the mid line or further, owing to the presence of a mesonephron

**Clinical Findings** The patient is usually a woman, and half the cases seen are between the ages of 30 and 40 The right kidney is affected at least twelve times as frequently as the left In many instances a movable kidney is found on routine examination without the patient being aware of any discomfort In other cases the patient complains of lassitude, aching, dragging or tenderness in the loin, flatulence, constipation or general nervous symptoms A special symptom, known as Dietl's crisis, may also occur This consists of an attack of severe pain in the lumbar region which radiates along the course of the ureter to the groin and inner side of the thigh The temperature falls and the pulse is frequent Micturition is scanty and there may be blood in the urine It is probably due to kinking of the ureter An intermittent hydronephrosis may also develop the renal capsule being distended owing to retention of urine in the pelvis, relief being obtained when the urine is evacuated

**On Examination** The patient is usually thin, with a narrow costal angle and lax abdominal wall Palpation of the kidney may produce nausea Varying degrees of renal mobility, as described above, may be detected

**Differential Diagnosis.** A palpable kidney on the right side must be differentiated from a Riedel's lobe of the liver (see p 73), an enlarged gall bladder, a pyloric or colonic tumour, and on the left side from an enlarged spleen or growth in the stomach or colon There is usually little difficulty X ray examination and pyclography serve also to demonstrate the size and position of the kidney. Dietl's crises simulate renal colic, and X ray examination and pyclography are of service in indicating the presence of a calculus

**Course and Complications** Generalised visceroptosis is often present

Complications include hydronephrosis, calculus formation, pyelitis, and adhesions between the kidney and duodenum. Jaundice may result from obstruction of the bile duct by adhesions.

**Prognosis.** This is good as regards the effect of movable kidney on life, but it is a cause of persistent ill-health.

**Treatment.** The patient should not be informed if a movable kidney, which is causing no symptoms, is discovered during a routine examination. If it is associated with general visceroptosis, the treatment for visceroptosis should be given (see p. 66), and a belt with a kidney support may be worn. An operation may be necessary to relieve intermittent hydronephrosis or Dietl's crises.

### Congestion of the Kidneys

Passive congestion occurs in heart failure (cardiac kidney) or is due to obstruction of the renal veins. The urine is diminished in volume, the specific gravity is raised, albumin is present and red cells, granular and hyaline casts may be found in the deposit.

### Infarction of the Kidneys

The emboli are usually derived from the heart. Minute bacterial emboli, in subacute infective endocarditis, give rise to focal nephritis (see p. 450). Larger infarcts cause severe pain in the kidney region with hæmaturia.

### Syphilis of the Kidneys

Chronic nephrosis may occur in the secondary stage (see p. 457). Later, gummata may form, with an associated amyloid degeneration of the kidneys.

### Renal Tumours

**Simple Tumours.** These are comparatively rare. They include a fibroma, adenoma, lipoma, papilloma and angioma. An angioma of the renal pelvis may give rise to severe and so-called "essential" hæmaturia. A papilloma of the pelvis tends to be locally malignant, recurring after removal and spreading down the ureter.

**Malignant Tumours.** These may be primary, and include a carcinoma, such as an adenocarcinoma (hypernephroma or Grawitz tumour). A squamous epithelioma may form in the renal pelvis. The primary sarcoma includes an embryoma (Wilm's tumour, containing muscles fibres and rarely cartilage and bone). Secondary carcinoma and secondary melanotic sarcoma also occur.

**Pathology.** The hypernephroma. This is now usually considered to be a papillary adenocarcinoma arising in the renal tubules. It may occur in any part of the kidney. It shows yellowish fatty areas, and hæmorrhages or cysts may be present. Secondary deposits form in the long bones, such as the tibia, in the bodies of the vertebræ, and in other organs, such as the lungs, liver and brain. The bony deposits are often very vascular. Other varieties of carcinoma include an alveolar adenocarcinoma and a tubular adenocarcinoma.

**Clinical Findings** The patient may be an infant, in which case the tumour is usually a sarcoma or hypernephroma

**On Examination** The child is weak, pale, wasted and the abdomen is swollen. A renal tumour may be present. The characteristic signs of this are: A tumour is felt in the loin, which enlarges downwards and backwards and later extends towards the mid line. It is dull on percussion, the dulness extending backwards to the flank. A band of colonic resonance may be found running across it. The tumour has a rounded border and the upper pole may be palpable. There is very slight movement with respiration. The urine may contain blood or albumin.

In an adult a hypernephroma may be so small that it gives rise to no local signs, or it may form a definite swelling. Pyelograms may reveal that one kidney is abnormal. Painless hæmaturia is the earliest sign of a renal tumour in the majority of cases. In the male the presence of a varicocele occurring for the first time in a patient of middle age, which does not disappear on lying down, is very suggestive of a malignant renal tumour especially if it is right sided.

If a secondary deposit forms in the tibia, a reddish brown discoloration of the skin may be seen over it, then a bony swelling is felt, and as this enlarges pulsation may be detected. If blood is present in the urine catheterisation of the ureters will show from which kidney it is coming.

**Prognosis** Death usually occurs in from 1 to 2 years from the date of diagnosis.

**Treatment** The results of nephrectomy are disappointing.

### Renal Cysts

The following varieties of cysts may occur in the kidneys: Congenital cystic kidney, Solitary cysts, Retention cysts, Hydatid cysts, Degeneration cysts in new growths.

### Congenital Cystic Disease of the Kidneys

#### (Polycystic Disease)

**Etiology** Congenital cystic disease is thought to be due to a faulty union of the renal tubules at the junction of those developed from the metanephros and those derived from the Wolffian duct. This point is situated in the collecting tubules. With an obstruction at this position a gradual dilatation of the tubules above occurs, with cyst formation.

**Pathology** The kidney may be considerably enlarged so much so that when occurring in the *fœtus* it causes difficulty in labour. The kidney substance may be almost entirely replaced by cysts. They contain a clear or turbid fluid, in which urea and blood may be present. One or both kidneys may be affected. In addition cysts may be found in the liver and pancreas and at times in the lungs.

**Clinical Findings.** The infant may be still born, or may die rapidly from uræmia. If occurring in adults no symptoms are usually noted.

until after the age of 30. The symptoms are then those of malignant renal sclerosis or of a renal tumour. The patient complains of lassitude, headaches, attacks of nausea and vomiting, or of an aching in the loin.

*On Examination:* An irregular renal tumour may be felt on one or both sides. The heart is usually enlarged, the arteries thickened and blood pressure raised. The urine resembles that of renal sclerosis, the volume being increased, the specific gravity low, and a trace of albumin being found, with an occasional hyaline or granular cast. Periodical attacks of hæmaturia may occur. As the lesion progresses, the blood shows retention of urea and non-protein nitrogen.

*Differential Diagnosis.* The diagnosis cannot usually be established unless the irregular knobbly kidney is palpable. An operation may be performed for a renal tumour, and the true nature of the lesion may only be discovered in this way.

*Course and Complications.* The course is progressive, with gradual failure of renal function. Polycystic degeneration of the liver and pancreas follows in some cases. Cerebral hæmorrhage may occur as a complication.

*Prognosis.* Death may ensue in from 3 to 25 years after congenital cystic disease has been diagnosed. The patient may live for many years with a high blood nitrogen content.

*Treatment.* This is as for renal sclerosis. The kidney should not be removed by operation.

### The Solitary Cyst

This is usually met with in children. It is probably a variety of polycystic disease. The cyst is generally situated in the renal cortex, and may contain over 2 pints of fluid. In adults large solitary serous cysts occur in the parenchyma of the kidney. They contain clear yellow fluid.

### Retention Cysts

These are met with post-mortem in cases of chronic nephritis. Clinically they give rise to no symptoms.

### Hydatid Cyst

This is due to an echinococcus infection. If the cyst ruptures into the renal pelvis, there is renal colic, and hooklets are found in the urine. It may also burst into the intestine or peritoneal cavity.

### Cystic Degeneration of a New Growth



## CHAPTER VI

### THE HÆMPOIETIC SYSTEM

**Introductory** The hæmopoietic system is concerned with the production during life of the formed elements of the blood. According to the polyphyletic view the red cells and granulocytes are produced by the bone marrow, the lymphocytes develop in lymph glands and lymphatic tissues generally, and the monocytes are derived from the reticulo endothelial system. The platelets are derived from megakaryocytes in the bone marrow. Certain factors are required for the maturation of the red cells. The primitive marrow cell is converted into a megaloblast possibly with the aid of an unknown agent. The active principle from the liver and stomach and perhaps the vitamin B complex (see p. 457) aid in the change from megaloblast to normoblast. Iron, copper, thyroxine and possibly vitamin C are concerned with the change from normoblast to erythrocyte. The reticulo endothelial cells are branched connective tissue cells with affinity for special dyes such as pyrrhol blue. They are widely distributed in the spleen, liver, bone marrow, omentum, adrenals, the pituitary, etc. Red cell destruction is effected by the reticulo endothelial cells in the spleen and liver, and possibly elsewhere. The total blood volume is approximately 5 litres.

The following examinations of the blood or bone marrow may be required in the investigation and treatment of diseases in this group.

1. *A Blood Count* A normal blood count for an adult is as follows. Red cells, average for both sexes, 5 000 000 per c mm. Hb average normal for males and females Haldane standard 105% Sahli standard 85%. These figures are equivalent to 14.5 G Hb per 100 c c blood. The size, shape and staining properties of the red cells are noted. Normally no nucleated red cells are seen, except in an infant for a few days after birth. Reticulocytes are immature red cells showing with vital stains a reticulum in their cytoplasm and normally present to the extent of  $\frac{1}{2}$  to 1%. They are the same cells as exhibit polychromasia when stained in a dry film. An excess of reticulocytes in the blood indicates that the red cells are being rapidly put forth into the circulation. The hæmoglobin content of each average red cell is indicated by (a) *The Mean Corpuscular Hb*. This is the weight of Hb in each average cell. The normal mean is 29.5  $\gamma$  ( $\gamma = 0.000000001$  mg). (b) *The Mean Corpuscular Hb Concentration*. This indicates the degree to which each red cell is saturated with Hb. The normal mean is 35%. (c) *The Colour Index*. This indicates the amount of Hb in each average red cell, and taking as arbitrary normal figures, Hb 100% and red cells 5 millions, the C I is  $\frac{\text{Hb \%}}{\text{Red Cells \%}}$ . The normal figure is 1. (d) *The Saturation Index*. This indicates the degree to which each average red

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The following examinations of the blood or bone marrow may be required in the investigation and treatment of diseases in this group.

1. *A Blood Count* A normal blood count for an adult is as follows. Red cells, average for both sexes, 5 000,000 per c mm. Hb average normal for males and females, Haldane standard 105%, Sahli standard 85%. These figures are equivalent to 14.5 G. Hb per 100 c c blood. The size, shape and staining properties of the red cells are noted. Normally no nucleated red cells are seen, except in an infant for a few days after birth. Reticulocytes are immature red cells showing with vital stains a reticulum in their cytoplasm, and normally present to the extent of  $\frac{1}{4}$  to 1%. They are the same cells as exhibit polychromasia when stained in a dry film. An excess of reticulocytes in the blood indicates that the red cells are being rapidly put forth into the circulation. The hæmoglobin content of each average red cell is indicated by (a) *The Mean Corpuscular Hb*. This is the weight of Hb in each average cell. The normal mean is 29  $\gamma\gamma$  ( $\gamma\gamma = 0.000000001$  mg). (b) *The Mean Corpuscular Hb Concentration*. This indicates the degree to which each red cell is saturated with Hb. The normal mean is 35%. (c) *The Colour Index*. This indicates the amount of Hb in each average red cell, and taking as arbitrary normal figures, Hb 100%, and red cells 5 millions, the C I is  $\frac{\text{Hb \%}}{\text{Red Cells \%}}$ . The normal figure is 1. (d) *The Saturation Index*. This indicates the degree to which each average red

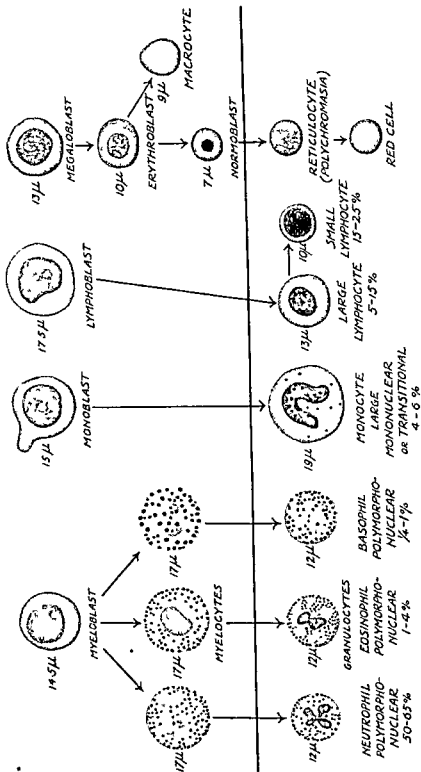


FIG. 46 THE BLOOD CELLS AND THEIR PRECURSORS ( $\times 1000$ ) THE CELLS BELOW THE LINE ARE NORMALLY PRESENT IN BLOOD

cell is saturated with Hb., as shown by comparing the mean corpuscular Hb. concentration of the blood in question with the normal mean corpuscular Hb. The normal saturation index is 1.

The size of each average red cell is indicated by: (a) *The Mean Corpuscular Diameter*. The normal average is  $7.2\mu$ . (b) *The Mean Corpuscular Volume*. The normal average is  $87\text{c}\mu$ . (c) *The Volume Index*. This indicates the volume of an average red cell of the blood in question compared with the normal average red cell volume. The normal figure is 1. The average life of a red cell is about 30 days; it is destroyed by disintegration. White cells: 6,000 to 9,000 per c.mm. Differential white count. Granulocytes. These include: Polymorphonuclears (neutrophils) 50 to 65%, eosinophils 1 to 4%, mast cells (basophils)  $\frac{1}{4}$  to 1%. Lymphocytes (small) 15 to 25%. Lymphocytes (large) 5 to 15%. Large mononuclears (monocytes, hyaline or transitional cells) 4 to 6% (see Fig. 46). The average life of a polymorphonuclear cell is four days, and of a lymphocyte less than a day.

2. *A Platelet Count*. Normally there are 200,000 to 500,000 platelets per c.mm.

3. *Fragility of the Red Cells*. Normally hæmolysis does not occur in solutions of sodium chloride, until the percentage of NaCl has been lowered from that of normal saline (0.9%) to a strength of 0.45%. With increased fragility hæmolysis may begin at 0.65% NaCl and be complete at 0.5%.

4. *The Bleeding Time*. A small cut is made in the ear or finger. Drops of blood are taken up on absorbent paper every 30 seconds, until bleeding stops. The normal time is 1 to 5 minutes.

5. *The Coagulation Time*. This varies with the method used, and a control should always be made to allow a normal reading as a comparison. The normal time by Dale and Laidlaw's method is  $1\frac{1}{2}$  to 2 minutes.

6. *The Sedimentation Rate of the Red Cells*. By the Westergren method, in which the tube is divided into 200 mm., the average normal reading at one hour is plasma 4 mm. (= plasma 2 mm. %) for men, and for women plasma 5.5 mm. (= plasma 2.7 mm. %). By Wintrobe's method the average normal sedimentation reading at one hour is plasma 3.7 mm., (= plasma 3.7 mm. %) for men, and for women plasma 9.6 mm. (= plasma 9.6 mm. %).

7. *The Estimation of Prothrombin in Plasma*. The clotting time of oxalated plasma, when mixed with an excess of thrombokinas and an optimum amount of calcium, is employed as a direct measure of the prothrombin content of the plasma. Using the method of Quick, normal plasma (100% prothrombin) should clot in 12 to 13 seconds.

8. *Sternal Puncture*. 0.25 c.c. of marrow fluid is removed by puncture of the sternum, using a Salah needle and dry syringe. The fluid is put in a tube containing Wintrobe's dry oxalate mixture, smears are then stained by Leishman's method.

9. *Blood Grouping*. The red cells may contain agglutinogens A or B, or both, or none; and the serum may contain agglutinins  $\alpha$  or  $\beta$ , or both, or none. Agglutinin  $\alpha$  or  $\beta$  causes agglutination of corpuscles

containing agglutinin A or B, respectively Four groups are described, according to the corpuscular agglutinin content

Group	Corpuscular agglutinin	Serum agglutinin
A, B or I	A B	O
A or 2	A	$\beta$
B or 3	B	$\alpha$
O or 4	O	$\alpha + \beta$

*Donors* Group AB may give blood to Group AB, Group A to A or AB, Group B to B or AB, and Group O is a universal donor

*Recipients* Group AB is a universal recipient Group A may receive blood from A or O Group B from B or O, and Group O from O It is better to use the blood of a donor of the same group as the recipient, rather than that of a universal donor Further, the serum of the recipient should always be tested against the corpuscles of the donor

In the investigation of certain cases of anæmia other tests are required, such as a fractional test meal, barium meal, the van den Bergh reaction the Wassermann reaction an examination of the urine for bile pigments and a test for occult blood in the feces

Various abnormal cells may appear in the blood in disease  
 1 *Abnormal red cells* Macrocytes are large non nucleated cells Microcytes are small non nucleated cells Megaloblasts and erythroblasts are large nucleated cells Normoblasts are normal sized nucleated cells The red cells may exhibit polychromasia (staining diffusely a bluish colour) or punctate basophilia (showing blue granules) They may be irregular in size (anisocytosis) or in shape (poikilocytosis and sickle cells)  
 2 *Abnormal white cells* Precursors of granulocytes These are premyelocytes with oval nuclei myelocytes with indented nuclei and metamyelocytes with a lobed nucleus These cells may contain neutrophil eosinophil or basophil granules and are classed accordingly The parent cell of the myelocyte is called a myeloblast, and that of the lymphocyte is a lymphoblast (see Fig 46) It is often very difficult to distinguish between a large immature lymphocyte (lymphoblast) and a myeloblast The myeloblast is slightly smaller, and often the oxidase test shows that the cytoplasm is granular In the very early stages however, the oxidase test is negative The oxidase test is negative with the lymphoblast the cytoplasm being agranular

### Leucocytosis

*Definition* An increase in the number of white cells in the blood

*Etiology* Physiological leucocytosis occurs after meals, exercise, cold baths and during labour and the first week of the puerperium In children there is a physiological lymphatic leucocytosis In pathological leucocytosis the different types of white cell may be unequally affected. *Polymorphonuclear leucocytosis* This is met with in septic

infections, in lobar pneumonia, in paroxysmal tachycardia, in coronary occlusion, in diabetic coma and after an acute hæmorrhage. 90% of the white cells may be neutrophil polymorphonuclears. *Lymphatic leucocytosis*: This occurs in lymphatic leukæmia, glandular fever, whooping-cough and sometimes in mumps. Up to 90% of the white cells may be lymphocytes. *Eosinophilia*: This is found in asthma, in parasitic infections such as ankylostomiasis and hydatid disease, in skin diseases such as psoriasis, urticaria and dermatitis herpetiformis, in polymyositis, and at times in acute polyarteritis nodosa, Hodgkin's disease or leukæmia, in chronic abdominal tuberculosis (see p. 186), or it may occur as a familial condition of unknown etiology. Up to 70% of the white cells may be eosinophils. A condition of eosinophilia with splenomegaly is described, due to syphilis, Hodgkin's disease, malaria or some unknown cause. *The mast cells* may be increased to about 20% in myeloid leukæmia. *The large mononuclears* may increase in infections such as malaria, syphilis or trypanosomiasis.

### Leucopenia

**Definition.** A diminution in the number of white cells in the blood.

**Etiology.** *Physiological leucopenia*: This occurs in old age and in starvation. *Pathological leucopenia*: This may be met with in tuberculosis, influenza, typhoid fever, pernicious anæmia, splenic anæmia, Hodgkin's disease, after exposure to radium, and in agranulocytic angina.

### Thrombocytosis

A temporary increase in the platelet count occurs after a hæmorrhage, childbirth and splenectomy. It is met with in polycythæmia vera, chronic myeloid leukæmia, Hodgkin's disease, in the remissions of pernicious anæmia, and in the anæmia of chronic sepsis, tuberculosis, carcinoma, etc. Thrombocythæmia is described on p. 523.

## ANÆMIA

The classification of the anæmias remains a most difficult problem, owing to the incompleteness of our knowledge of their etiology. For clinical purposes anæmias were formerly grouped as primary and secondary. The cause of the primary anæmias was unknown, whereas secondary anæmias were due to some recognisable agent, such as infection or hæmorrhage. As in all cases anæmia must result from a deficient formation of red cells or from their excessive destruction, this classification becomes more unsatisfactory as our knowledge increases. Wintrobe, in 1930, grouped some of the anæmias according to the size and hæmoglobin content of the red cells. It should be realised that although a red cell may contain less hæmoglobin than normal in proportion to its size (hypochromic anæmia), the proportional amount of hæmoglobin is never increased. There is therefore no such condition as hyperchromic anæmia, the high colour index in pernicious anæmia being due to large red cells containing a normal percentage of hæmoglobin. Wintrobe's classification is as follows: 1. *Macrocytic anæmia*,

as in pernicious anæmia, achrestic anæmia, the anæmia associated with liver disorders such as cirrhosis, passive congestion, carcinoma, and acute yellow atrophy, and with myxœdema, the anæmia of sprue, tropical macrocytic anæmia, the pernicious anæmia of pregnancy, the anæmia of chylous diarrhœa and of *Diphyllobothrium latum* infestation.

2 *Normocytic anæmia*, as in aplastic anæmia, malaria and after an acute hæmorrhage. 3 *Simple macrocytic anæmia*, as in chronic infections and carcinoma. 4 *Microcytic hypochromic anæmia*, as in chronic hæmorrhage, infestation with hookworms, simple achlorhydric anæmia, the Plummer-Vinson syndrome, chlorosis and the simple nutritional anæmia of infants. A definite advantage of Wintrobe's classification is that it emphasises the distinction between macrocytic and microcytic hypochromic anæmias. This is of clinical importance, as, if the red cells are saturated with hæmoglobin, the macrocytic anæmias are benefited by liver therapy, and the hypochromic anæmias by iron. Iron is, however, required for macrocytic anæmias, if the saturation index is low.

Davidson, in 1932, put forward a classification with an etiological basis. This is obviously sound, but there are at present gaps in our knowledge which render it incomplete. The following classification is that of Davidson with some modifications.

1 *Nutritional deficiency anæmias*. These may be due to: (a) Lack of the anti-anæmic principle (see p. 187). This occurs as a primary defect (the diet being adequate) in pernicious anæmia, and as a secondary defect (*i.e.*, resulting from some recognisable cause) in the anæmia of sprue, dysentery and tropical megalocytic anæmia, in the pernicious anæmia of pregnancy, in *Diphyllobothrium latum* infestation, in carcinoma of the stomach and after gastrectomy. In this latter group there is also often an error in iron assimilation. There may be a deficiency of storage of the hæmopoietic principle in advanced cirrhosis of the liver. (b) Lack of the factors required for hæmoglobin formation such as iron and possibly copper, calcium, thyroxine, vitamin C, chlorophyll, and a salt balance in the food. This also may be a primary defect, as in the simple achlorhydric anæmia and the Plummer-Vinson syndrome where the food intake is often satisfactory, or secondary to a recognisable food or endocrine defect, as in the simple nutritional anæmia of infants, chlorosis, chronic gastritis and enteritis, cœliac disease, starvation, prolonged milk feeding as in the treatment of peptic ulcers, and in myxœdema and thyrotoxicosis.

2 *Post-hæmorrhagic anæmias*. These occur after acute or chronic hæmorrhage.

3 *Hæmolytic anæmias*. (a) Acute hæmolysis may result from black-water fever, malaria, paroxysmal hæmoglobinuria, septicæmia, or toxins, such as snake venom. It also occurs in Lederer's anæmia. (b) A persistent hæmolysis, as in congenital or acquired acholuric jaundice, sickle-cell anæmia, Cooley's anæmia, Von Jaksch's anæmia, and lead poisoning.

4 *Anæmias due to inhibition of the bone marrow function*. These may be primary, as in aplastic anæmia, or secondary, as in aplastic anæmia due to noxious agents such as X rays, radium emanations, benzol, lead or mercury. A leuco-erythroblastic anæmia

occurs owing to involvement of bone marrow in carcinomatosis, myelomatosis and in osteosclerosis (Marble-bone disease of Albers-Schönberg).

By some writers groups 1 and 4 in the above classification are grouped together as the *Dyshæmopoietic anæmias*.

## NUTRITIONAL DEFICIENCY ANÆMIAS

### Pernicious Anæmia

#### (Addisonian Anæmia)

**Definition.** A severe anæmia of uncertain origin, characterised by a typical blood picture, and a progressive course which is often interrupted by remissions.

**Etiology.** The accepted theory is that pernicious anæmia results from the lack of a hæmopoietic or anti-anæmic principle, known also as the pernicious anæmia (P.A.) factor. This hæmopoietic principle is formed from the interaction of an extrinsic and an intrinsic factor in the small intestine. The "extrinsic factor" is present in protein foods. The "intrinsic factor" of Castle occurs in normal gastric juice, and is probably a rennin-like ferment. It is also known as "hæmopoietin" (Wilkinson). Meulengracht believes that in the pig the "intrinsic factor" is secreted by the pyloric glands and by Brunner's glands in the duodenum. These two sets of glands constitute the "pyloric gland organ." Pepsin and hydrochloric acid, on the other hand, are formed in the fundus of the stomach. The hæmopoietic principle thus formed is carried to the liver and other organs, and stored in the liver, and to a lesser degree in the kidneys and spleen. In its absence pernicious anæmia develops owing to the failure of conversion of megaloblasts into erythroblasts and normoblasts in the bone marrow. When these megaloblasts are liberated into the blood stream they are destroyed by the cells of the reticulo-endothelial system. The iron which is deposited in the spleen, liver and kidneys, and the excess of bilirubin in the blood, are in part due to this hæmolytic, and in part to the failure of the bone marrow to utilise these substances in the production of red cells.

In pernicious anæmia the hæmopoietic factor is not formed, owing to the absence of the "intrinsic factor" from the gastric juice. This is associated with achylia. Achylia gastrica has been demonstrated in the majority of cases, the gastric juice showing no free HCl, pepsin or rennin after the intramuscular injection of 1 mil. of 0.1% solution of histamine. If gastric juice obtained from a patient suffering from pernicious anæmia is incubated with beef, the hæmopoietic principle is not produced, owing to the absence of the "intrinsic factor." Sturgis and Isaacs have also shown that the hæmopoietic principle is present in dried whole hog's stomach. Wilkinson demonstrated that the mucous membrane and muscle layer of the hog's stomach yield a light powder when desiccated at 40° C. *in vacuo*. This stomach "extract" is potent in the treatment of pernicious anæmia. The "extrinsic factor" is possibly closely allied to the vitamin B complex.



Marmite a yeast extract, which contains vitamin B<sub>1</sub> and nicotinic acid, is in some cases efficacious in the treatment of pernicious anæmia, but probably only in those cases in which there is a small quantity of the intrinsic factor present in the gastric juice

*Predisposing causes* 1 Age Usually after 35 years 2 Sex No definite difference 3 A familial incidence is noted in some cases 4 Extensive gastrectomy 30% of such cases may develop pernicious anæmia

*Pathology* Post mortem the body is well nourished The skin has a lemon yellow tint the subcutaneous fat is yellow, and the muscles rather dark red The heart is soft owing to fatty degeneration and tabby cat striation is seen under the endocardium and on the musculi papillares and columnæ carneæ The tongue is smooth and may show ulcers on its edges The mucous membrane of the stomach is atrophied and petechial hæmorrhages may be seen Microscopical examination shows changes in the fundus with atrophy of glands and disappearance of the parietal and chief cells but the glands in the pylorus are well preserved and no changes are seen in Brunner's glands In man as opposed to the pig it appears that the intrinsic factor is formed in the cardia and body of the stomach The liver is slightly enlarged owing to fatty infiltration The spleen is usually enlarged, but may be smaller than normal Owing to the deposition of free iron (hæmosiderin) a prussian blue reaction is given by sections of the liver, and occasionally by the spleen and kidneys, on applying a 2 to 5% solution of potassium ferrocyanide followed after washing by 5 to 10% hydrochloric acid The red marrow of the bones is increased, especially in long bones such as the femur Degeneration may be found in the postero lateral columns of the cord

*Clinical Findings* The patient is usually an adult between the ages of 35 and 45 years Very rarely pernicious anæmia occurs in children He complains of progressive weakness increasing pallor with dyspnoea on exertion palpitations and at times anginal pains due to anoxia of the heart muscle In some cases swelling of the feet or ankles may be noted There may be soreness of the tongue numbness or tingling in the legs and hands vomiting or diarrhoea Failing vision, due to optic atrophy may be the first symptom

*On Examination* The nutrition of the patient is usually good The tongue may be very smooth. The skin is pale with at times a lemon yellow tint There may be cutaneous pigmentation with areas of leucoderma and small petechiae may be seen The spleen and liver are often just palpable Evidence of postero lateral spinal sclerosis may be found such as patchy anæsthesia of the legs, weakness of muscles and an extensor plantar response. Cases in which there is tenderness of the calves weakness of the legs tingling numbness, and depressed tendon reflexes may be suffering from peripheral neuritis or an early stage of subacute combined degeneration In some instances there is definite jaundice and ascites The blood A typical count during a relapse phase is as follows Red cells 500 000 to 2 500 000 per c.mm. Hb, 12 to 65% C I, 11 Mean corpuscular Hb 50% Mean corpuscular

Hb. concentration 83%. Saturation index 1. The average size of the red cells is  $8.24\mu$  (normal  $7.2\mu$ ). Mean corpuscular volume  $150\text{ cu.}$  Volume index 1.5. The red cells show anisocytosis, poikilocytosis, megalocytosis, polychromasia and punctate basophilia. Normoblasts and megaloblasts are present. The platelets are reduced. Reticulocytes are increased to about 2%. White cells: There is a leucopenia, 4,000 to 5,000 per c.mm., with relative lymphocytosis up to 50%. The polymorphonuclears show a "shift to the right" in the Arneth count, many having a four or five lobed nucleus. There are usually some myelocytes present. The coagulation time: This is prolonged. The sedimentation rate: This is increased, owing to the anæmia alone and not to tissue destruction. The serum: An indirect van den Bergh reaction is given. The bone marrow obtained by sternal puncture in untreated cases shows 25 to 45% of the cells to be megaloblasts and erythroblasts, and premature hæmoglobinisation is present. The urine and fæces contain an excess of bilirubin. During treatment with liver an output of reticulocytes occurs before the number of red cells increases, and there may be an eosinophilia up to 26%, during the eighth to twelfth weeks of raw liver treatment. The fractional test meal shows achylia gastrica, no free HCl being secreted after injection of histamine (see p. 487). The juice also lacks pepsin and the "intrinsic factor" (see p. 487). Rarely the juice contains acid and pepsin, but the "intrinsic factor" is absent. The stomach empties rapidly.

**Differential Diagnosis.** The diagnosis of pernicious anæmia depends upon the typical blood count, with megalocytosis, high colour index, and the presence of large nucleated red cells, and the fractional test meal showing an achylia and absence of the "intrinsic factor" (see p. 487). During the remission phases, although nucleated red cells may be absent in the blood film, yet the average size of the red cells remains greater than normal. Difficulty may arise with severe cases of septic anæmia, with a high colour index, but the average red cell size is not increased. In carcinoma of the stomach (see p. 41) the blood count may closely resemble that of pernicious anæmia. The X-ray findings and occult blood test are usually helpful. In sprue (see p. 692) and infections with the *Diphyllobothrium latum* (see p. 713) the blood picture may be similar to that of pernicious anæmia, but achylia is not present. If there is much pigmentation Addison's disease may be suspected. The blood count serves to differentiate. Postero-lateral sclerosis may occur before the development of the typical blood picture of pernicious anæmia. Achrestic anæmia ( $\chi\rho\eta\sigma\theta\alpha\iota$  = to use) is described by Wilkinson and Israëls. The anæmia is megalocytic, the patient being unable to use the hæmopoietic principle. Free HCl is present in the gastric juice and there are no complications. It is resistant to liver therapy.

**Course and Complications.** Remissions are common, during which the patient feels and is better, and the blood count improves. Blood crises may also be noted, characterised by the appearance of large numbers of nucleated red cells. Some cases pursue a course rapidly fatal in a few days or weeks, in others life is prolonged for 2 or 3 years.

It is doubtful if permanent recovery, apart from treatment, ever occurs. Even after successful treatment the achylia nearly always remains. Complications include trophic ulcers on the buttocks and heels, pneumonia and nephritis.

**Prognosis** The disease is fatal usually within two to three years, if no treatment is given. The introduction of the liver and stomach extract treatment has revolutionised the outlook, and it is now very rare for a patient to die during the acute stage if adequate treatment is given. Although the death rate from pernicious anæmia fell in 1928; following the introduction of liver treatment the previous year, the death rate has been rising again since 1931. This may be due to inadequate treatment, to the patient becoming refractory to treatment, or to faulty certification, the patient dying from some other disease.

**Treatment** The introduction of the Murphy Minot liver treatment constituted an advance of the first magnitude in medicine.

Originally 250 G of raw or lightly cooked liver of the ox, calf or pig were ingested daily. Now a liver extract is usually employed. In a severe case, with red cells below 1 million per c mm, the patient is put to bed and an intravenous injection of a preparation such as Hepatex PAF is given very slowly, the dose being 5 mls diluted in 20 mls of warm normal saline solution. An initial blood transfusion is now seldom required. Alternatively 6 to 8 mls of Hepatex IM, or Hepastab are injected intramuscularly. The next day 4 mls are injected intramuscularly and this dose is repeated daily until a reticulocyte crisis occurs. This is to be expected between the fourth and tenth days. The peak of the reticulocyte rise varies inversely with the original degree of anæmia. Thus with an initial red cell count of 0.5 million, the reticulocytes may touch 55%, with 1 million red cells a reticulocyte figure of 35% may be expected, with 2 million red cells 14% reticulocytes, and with 3 million red cells 4% reticulocytes. The number of reticulocytes then falls rapidly by a crisis as the mature red cells start to increase in the blood. Subsequently 2 to 4 mls of the liver preparation are injected intramuscularly every week until the blood count is normal and there are no symptoms. Then a maintenance dose of 2 mls is required every two, three, four, or even six weeks. If the blood count does not rise over 3 or 3.5 millions or the mean corpuscular Hb concentration is below 30%, iron should also be given as ferri cit ammon cit in doses of gr 30 to 45 t i d, and iron is especially valuable in all cases in which nervous symptoms are present. Highly purified liver products, such as Anabamin, should not be used in these cases. The great advantage of treating patients by injection of liver concentrates lies in the fact that the patient must report every three or four weeks for the injection, when a blood count can be done to ensure that the hæmoglobin is maintained at over 90% and the red cells over 4 millions. Failure to do this will involve the patient in the risk of developing postero lateral sclerosis and becoming a cripple.

Some authorities prefer to treat pernicious anæmia by desiccated preparations of hog's stomach administered orally. Thus Extomak, Pepsac,<sup>1</sup> and Ventriculin are available, the dose being 10 G t i d. The

powder must be given cold on food or in drinks. The maintenance dose is usually 28 G. daily. A more concentrated liver preparation, Anahæmin, is put up in ampoules containing 100 mg. in 1 mil. In a severe case 0.5 mil. should be injected daily for a week, the reticulocyte crisis being expected between the fourth and seventh days, or a single injection of 4 mils may be given intramuscularly. At the end of a week, if a response has been obtained, 2 mils should be injected intramuscularly, and repeated every two weeks until the blood count is normal. A maintenance dose of 2 mils every four weeks may be all that is required. As soon as the patient has recovered from the acute stage of the disease, any septic focus in the mouth should be treated. Acid. hydrochlor. dil. m. 30 to 60, syr. aurant. m. 30, aq. ad fl. oz. 1. Fl. oz. 1 in 6 oz. of water with and after meals, t.i.d., will in some cases improve the appetite.

### The Pernicious Anæmia of Pregnancy

During pregnancy a macrocytic anæmia of a "pernicious" type may arise, as described by Osler. It differs from pernicious anæmia in its tendency to spontaneous recovery with the termination of pregnancy, and in the presence of a normal gastric acidity. There may be hæmorrhages from the nose, alimentary tract or the vagina. The deadly pallor, dyspnœa, tachycardia, fever, syncopal attacks, transient paralysis or even blindness suggest the occurrence of an internal hæmorrhage.

**Differential Diagnosis.** This anæmia must be differentiated from Addisonian pernicious anæmia complicated by pregnancy, from an aplastic or chlorotic (hypochromic) anæmia during pregnancy, and from an acute internal hæmorrhage.

**Treatment.** An immediate blood transfusion of 500 muls is required, followed by the administration of liver concentrate as for pernicious anæmia (see p. 490). It may be necessary to terminate the pregnancy in severe cases which have not received adequate treatment or which have not responded to it.

### The Anæmias of Sprue and of *Diphyllobothrium Latum* Infestation Tropical Megalocytic Anæmia

These anæmias are of the macrocytic type, but the gastric secretion is normal. The tropical megalocytic anæmia is probably due to a dietary deficiency. In *Diphyllobothrium latum* infestation there is thought to be a deficient absorption of the anti-anæmic principle.

**Treatment.** A good response is usually obtained with liver concentrates, provided they are not highly purified, or with Marmite,  $\frac{1}{2}$  oz. daily.

### Simple Achlorhydic Anæmia (Faber)

#### (*Idiopathic Microcytic Anæmia. Essential Hypochromic Anæmia*)

The patient is usually a woman of middle age. She complains of pallor, dyspnœa, swelling of the feet, palpitations, indigestion and, in a severe case, of anginal pain. The predominance in the female sex is probably due to uterine losses of blood. The food intake is often satisfactory but may be deficient in iron, or the iron is not adequately absorbed. Normally 15 mg. of iron per day are required.

**On Examination** The skin is pale and often sallow. The nails are concave or spoon shaped (koilonychia) in about 40% of cases and brittle (see Fig 47). Cracks are often seen at the corners of the mouth. The tongue is smooth, red, but not sore. The spleen may be enlarged. There are no changes in the spinal cord. The test meal shows a complete achlorhydria with rapid stomach emptying, and mucus is present in excess, there is usually some pepsin, and some HCl response to the intramuscular injection of histamine. Castle has shown that if beef is incubated with the gastric juice, the "haematine principle" of pernicious anaemia is produced even in specimens of histamine-refractory gastric secretions. This shows that the juice contains Castle's "intrinsic factor" (see p 187) and explains why the patient does not develop pernicious anaemia. Occasionally normal test meal findings are recorded. The van den Bergh test on the blood is negative, the faeces contain no occult blood. The blood count is that of a hypochromic microcytic anaemia with a low colour index, such as, red cells 3 to 4 millions per c mm, Hb 30 to 50% C.I. 0.4 to 0.5. The majority of the red cells are smaller than normal. Complications include the Plummer-Vinson syndrome (see p 17).

**Treatment** If possible the cause of the anaemia should be removed. It is well to give a scale preparation of iron such as *Ferri et ammon cit* in doses rising from gr 5 to gr 60 in a mixture with glycerin m 20, aq menth pip dest ad fl oz 1. Fl oz 1 t i d s p c. Alternatively *Blaud's pill* (*pil ferri carbonatis B.P.*) gr 15, freshly prepared and cut into two parts before being swallowed, t i d, or *Tab bersolate* gr 3, may be given. Each tablet contains ferrous sulph gr 1, copper sulph gr 1/100 and manganese sulph gr 1/100, and is equivalent to about gr 80 of *ferri et ammon cit*. In any case sufficient iron must be given to provoke a reticulocyte crisis. Acid hydrochlor dil m 20 to 40 t i d s p c in a glass of water should also be given in cases of achlorhydria. A blood transfusion may be required in very severe cases.

### The Plummer-Vinson Syndrome

This is described on p 17.

### The Nutritional Anaemia of Infancy

Mackay showed that 51% of artificially fed babies and 45% of breast fed babies in London of the hospital class were anaemic. This is due to deficiency of iron and minerals in milk, and is curable by the administration of iron. The following mixture is used. *Ferri et ammon cit*, gr 1½ aq chlorof m 60. Add 2 to 3 drops of the mixture to the feeds three times a day, and gradually increase up to m 60 t i d. The mother also is frequently anaemic.

### Chlorosis

#### (The Green Sickness)

**Definition** An anaemia of young girls, characterised by diminution of haemoglobin, excess of blood volume, and a favourable response to treatment with iron.

present in the blood which results in the formation of spherocytes. The spleen may destroy these abnormally thick and unduly fragile red cells. There is a familial incidence with an equal sex distribution. The symptoms are usually noted before the age of 10.

**Pathology.** The spleen is moderately enlarged, but the capsule is not usually thickened. The pulp contains many red cells, which are ingested and destroyed by the endothelial cells. The red bone marrow is hyperplastic.

**Clinical Findings.** Often no symptoms are noted, but the patient may complain of jaundice, or of attacks of vomiting, or of weakness and anæmia.

**On Examination :** There is some icterus of the skin and conjunctivæ. The spleen is enlarged. Long-standing ulceration of the legs may be noted. The urine is dark and contains urobilin, but no bile pigment. The blood : The red cells are fragile, undergoing hæmolysis in 0.65% NaCl solution, whereas normally hæmolysis does not occur until the strength of the NaCl is lowered to 0.45%. The increased fragility is probably associated with the spheroidal shape of the red cells, whereby their volume is not diminished. The average diameter of the red cells is reduced (microcytosis). There is some anæmia, but the colour index is generally just under unity. A few normoblasts and an excess of reticulocytes are present. The white cells are normal. Blood crises occur in which there is a more marked anæmia, a leucocytosis, and the number of reticulocytes is increased. The serum gives an indirect van den Bergh reaction, the jaundice being of a hæmolytic type. The fæces are dark and contain urobilin and urobilinogen. Other congenital defects such as oxycephaly may be present.

**Differential Diagnosis.** This is established by the familial incidence, the microcytosis with jaundice and the increased fragility of the red cells. A blood examination during a crisis may suggest a leukæmia or the acute hæmolytic anæmia of Lederer, owing to the leucocytosis with the presence of some primitive granulocytes.

**Course and Complications.** The disease does not necessarily produce any serious effects upon the patient's health, but an intercurrent septic infection may result in an "anæmic breakdown" later in life. Gallstones and deposits of urates around the joints may occur subsequently.

**Prognosis.** This is good with adequate treatment.

**Treatment.** Splenectomy will cure the condition as regards the jaundice and anæmia, although the fragility of the red cells after splenectomy is still greater than normal. It is only required in cases in which crises occur and should be performed in the remission stage, and a blood transfusion should not be given. In very severe crises splenectomy may be performed as an emergency measure after a small preliminary blood transfusion.

### Acquired Acholuric Jaundice

**Definition.** A variety of acholuric jaundice appearing in adults.

**Etiology.** The cause is often unknown. It may result from lesions, such as toluylene-diamine, or from infections such as malaria,

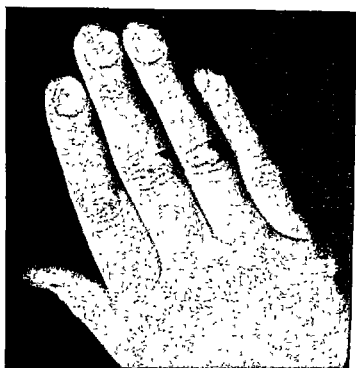


FIG. 47. SPOON-SHAPED NAILS (KOILONYCHIA) IN SIMPLE ACHLORHYDRIC ANEMIA.



FIG. 48. DILATED VEINS ON TRUNK AND ARMS RESULTING FROM OBSTRUCTION OF THE SUPERIOR VENA CAVA.

The patient, aged 12, was suffering from subacute lymphatic leukemia with a mediastinal leukæmic tumour. The first symptom was intermittent swelling of the face and neck, which disappeared as the collateral circulation was established.



syphilis, tuberculosis, or dysentery. It is by many authorities considered to be indistinguishable from the congenital variety, symptoms, however, being noted for the first time in adult life.

**Clinical Findings** The patient is an adult, often of the female sex. The onset is frequently sudden with nausea, vomiting and jaundice. Later, attacks of biliary colic may occur.

**On Examination** The spleen is enlarged. The blood. There is a hæmolytic anæmia, usually of a greater severity than in the congenital type. The red cells show a high mean corpuscular volume, a high *CI* and a high mean cell diameter, but the low mean corpuscular hæmoglobin is an important distinguishing point from the findings in pernicious anæmia. The fragility is often normal. The reticulocytes are usually increased, apart from treatment. The urine contains urobilin. Gall stones occur as a complication.

**Treatment** Splenectomy will sometimes effect a cure, but should not be performed until any possible cause of the disease has been treated. Blood transfusion is dangerous, as it may provoke a hæmolytic crisis.

### Sickle-cell Anæmia

#### (*Drepanocytic Anæmia*)

**Definition** A severe anæmia in which the red cells assume an elongated or sickle shape.

**Etiology** The cause is unknown, but it is thought to be a hæmolytic anæmia. Sickle-cell anæmia occurs chiefly in negroes of North America. Both sexes are affected, and there is a familial incidence. The patient is usually under 30 years of age.

**Pathology** The liver is usually enlarged, and there is hypertrophy of bone marrow and lymphoid tissue. The spleen is enlarged early in this disease, owing to congestion, but later, as the result of siderofibrosis, it is small. The 'sickling' depends upon the partial oxygen pressure. If the arm or finger is compressed, in order to reduce the oxygen supply, immediately before the blood is taken, the number of sickle cells seen in the direct film may be 90%.

**Clinical Findings** The patient complains of weakness, pains in the muscles and abdomen, vomiting, diarrhoea and irregular fever. Slight jaundice may be present. The blood. The red cells are diminished in number. The average size is slightly enlarged (Wintrobe). The sickle shape appearance of the red cells is only seen in blood which is freshly drawn and allowed to stand under a sealed cover slip. In about 12 to 24 hours over 60% of the red cells become elongated and return to their normal shape in another 24 to 48 hours. There is anæmia of varying degree, with some nucleated red cells, polychromasia and reticulocytosis. The colour index is below unity. There is a leucocytosis of 12 000 per c mm, or more. The fragility of the red cells is decreased and the indirect van den Bergh reaction is positive. 'Sickling' of the blood may also occur without any symptoms of ill health (sicklæmia).

**Prognosis.** Recovery rarely takes place, the patient usually developing an intercurrent infection before the age of 35.

**Treatment.** General hygienic measures should be adopted and a course of liver treatment tried, giving 8 oz. of liver or its equivalent of extract daily.

### Cooley's Anæmia

(*Erythroblastæmia of Childhood. Thalassæmia*)

**Definition.** A severe anæmia occurring in childhood with splenomegaly and characteristic bone changes. A benign form of Cooley's anæmia occurring in adults has also been described.

**Etiology.** The cause is unknown. It occurs chiefly in the Mediterranean areas, rarely in England and America. There is a familial incidence.

**Pathology.** There is hyperplasia of the bone marrow, with islands of megaloblasts. The spleen is fibrosed.

**Clinical Findings.** The patient is an infant or child who suffers from lassitude. The skin is yellowish or muddy in colour, the aspect somewhat Mongoloid, with thickening of the malar bones. The spleen and liver are enlarged. The blood: Red cells, 2 to 4 millions per c.mm. There is anisocytosis, poikilocytosis and many normoblasts. Hb. 80 to 60%. C.I. low. Fragility normal. White cells, 15,000 to 50,000 per c.mm. A few myeloid cells are seen in severe cases. Platelets normal. Indirect van den Bergh positive. X-ray examination shows changes in the skull, the long bones and the small bones of the hands and feet. There is rarefaction and trabeculation of the long bones. Radiating spicules are seen in the lateral view of the skull.

**Differential Diagnosis.** Cooley's anæmia is closely allied to von Jaksch's anæmia. Other conditions which require exclusion are acholuric jaundice, sickle-cell anæmia, leukæmia and syphilis.

**Course and Complications.** The disease is steadily progressive.

**Prognosis.** Death usually occurs before the age of 10.

**Treatment.** Temporary improvement may result from the administration of iron.

### Von Jaksch's Anæmia

(*Splenic Anæmia of Infants. Anæmia Pseudo-leukæmia Infantum*)

**Definition.** A disease of infants characterised by enlargement of the spleen, liver and lymph glands, a severe anæmia and leucocytosis. Some authorities consider that this condition is not a disease entity, but includes several varieties of nutritional anæmias.

**Etiology.** The cause is unknown. It is often associated with rickets, and so is more common in infants who are bottle-fed. Syphilis or malaria may be predisposing causes. It is also thought to be a subacute hæmolytic anæmia due to an infection.

**Pathology.** The spleen is enlarged and firm with a thickened capsule. Myeloid cells may be seen on microscopical examination. The bone marrow is red and hypertrophied. The lymph glands are enlarged. It is not known whether it is really a leukæmia.

**Clinical Findings.** The first symptoms are usually noted between the ages of 6 months and 3 years. There is marked pallor and muscular weakness, hæmorrhage may occur from the nose or into the skin. The spleen and liver, but not usually the lymph glands, are enlarged. The blood. Red cells, one million per c mm. or less. Hb 15 to 20%. C.I. 0.5 to 0.6. Normoblasts and megaloblasts are present, and there is poikilocytosis, polychromasia and punctate basophilia. White cells, 30,000 to 100,000 per c mm. There is lymphocytosis, with myelocytes and myeloblasts. Platelets are diminished.

**Differential Diagnosis.** The diagnosis is established by the clinical findings and the blood count. The fact that certain patients recover, and the absence of bone changes, other than those associated with rickets, distinguish von Jaksch's anæmia from Cooley's anæmia. The diagnosis from chronic leukaemia may be very obscure.

**Course and Complications.** The course is usually progressive. Complications include bronchopneumonia and gastro-enteritis.

**Prognosis.** A variable proportion of cases proves fatal, but recovery may occur.

**Treatment.** Rickets, syphilis or malaria if present, should be treated, and the child placed in good hygienic surroundings. In severe cases a blood transfusion should be given, followed by the administration of iron by mouth.

## ANÆMIAS DUE TO INHIBITION OF BONE MARROW FUNCTION

### Aplastic Anæmia

#### (*Leukia hæmorrhagica*)

**Definition.** A severe anæmia, closely resembling pernicious anæmia, but the course is not interrupted by remissions.

**Etiology.** Aplastic anæmia is classified as a primary anæmia, as in many cases no cause can be discovered. In some cases it is due to exposure to X rays or radium, or to poisoning with benzol, gold, phenylhydrazine, the sulphonamides, or mustard gas. It may also occur as a terminal phase of untreated pernicious anæmia, or of polycythæmia rubra, or be due to replacement of bone marrow by sarcomatous or carcinomatous tumours, or complicate acute infections especially in children, such as influenza and diphtheria.

**Pathology.** There is marked aplasia of the marrow of the long bones, the normal marrow being replaced by fat, and iron is not deposited in the internal organs.

**Clinical Findings.** The patient is usually a young adult of either sex, who notices weakness, pallor, dyspnoea and palpitations as in pernicious anæmia. The skin may have a yellow tinge. Purpura or hæmorrhages from mucous membranes, or from the uterus may occur. The blood. Red cells, 0.5 to 1.5 million per c mm. The cells are generally of normal appearance. There are usually no reticulocytes. Hb 10 to 30%. C.I., 0.9 to 1. White cells, 1,000 to 2,000 per c mm. There is a relative lymphocytosis (up to 60%). No myelocytes are seen. Platelets are diminished. Bone marrow removed by sternal

trephine is usually hypoplastic showing a lack of mature cells; occasionally it is hyperplastic with many primitive cells. Achlorhydria is not present. The van den Bergh reaction is negative.

**Differential Diagnosis.** The severe nature of the anæmia is apparent. The blood count distinguishes it from pernicious anæmia and agranulocytosis. In aleukæmic lymphadenosis (lymphæmia) and in aleukæmic myelosis (leukæmia) lymphoblasts or myeloblasts are present, and examination of the bone marrow serves to differentiate.

**Course and Complications.** The course is steadily progressive.

**Prognosis.** Death is to be expected in less than a year unless there is some cause which can be removed.

**Treatment.** The effect of liver treatment in this disease is usually disappointing. Iron and arsenic are without avail. A blood transfusion produces only temporary improvement, but repeated drip transfusions may keep the patient alive for several years. Very occasionally splenectomy is successful; more often the patient dies within a few days of the operation.

## THE LEUKÆMIAS

### (The Leucoses)

**Definition.** Progressive diseases of the hæmopoietic system, characterised by an increase in the white cells and their precursors in the blood and changes in the myeloid or lymphoid tissues of the body.

**Etiology.** The cause is unknown. It is possible that the changes are of the nature of a new growth or in some cases a response to infection.

**Varieties.** Leukæmia may be acute or chronic, and is subdivided further into acute lymphatic leukæmia (lymphadenosis), acute myeloid leukæmia (myelosis), acute monocytic (histiocytic) leukæmia, leukanæmia (panmyelosis), chloroma, chronic lymphatic leukæmia, and chronic myeloid leukæmia.

**Pathology.** The types of white cells which are seen in the blood in the leukæmias are largely embryonic, such as myelocytes, myeloblasts, lymphoblasts, etc. The only way to learn to recognise these cells is by having them demonstrated in blood films under the microscope, and a detailed description will not therefore be given (see Fig. 40).

### Acute Lymphatic, Acute Myeloid and Acute Monocytic Leukæmias

Clinically it is impossible to distinguish between these diseases, apart from the blood examinations. They are comparatively rare.

**Pathology.** *Acute Lymphatic Leukæmia.* There is hyperplasia of lymphatic tissue throughout the body, with enlargement of the lymph glands, and infiltration of the spleen, liver and bone marrow with lymphocytic cells.

*Acute Myeloid Leukæmia.* The lymph glands are enlarged to a varying degree, and the spleen, liver and bone marrow are infiltrated with myelocytes and premyelocytes.

*Acute Monocytic Leukæmia.* The spleen, liver, bone marrow and lymph glands are infiltrated with embryonic monocytes (histiocytes).

In all varieties hæmorrhages may be found in such organs as the

stomach, intestines, lungs, brain, kidneys, into the buccal mucous membrane and under the skin

**Clinical Findings** The patient is usually a male under the age of 20. Females are only affected half as frequently. The onset usually resembles a feverish attack or chill, or a hæmorrhage from the mouth, rectum, vagina or other site may be the initial symptom. Intractable hæmorrhage after dental extraction may be the first indication of the disease. The patient may complain of pain in the bones.

**On Examination** The patient looks pale and ill, and purpuric spots or larger ecchymoses may be seen in the skin. The temperature is irregular, between  $99^{\circ}\text{F}$  (m) and  $101^{\circ}\text{F}$  (e) or higher. The pulse is proportionately rapid. Necrotic lesions may be seen in the mouth or fauces. The liver or spleen may be just palpable and the glands in the neck, axillæ or groins enlarged. A mediastinal type occurs (see Fig 18, facing p. 497), in which pressure signs point to a mediastinal tumour, this is confirmed by X ray examination. The blood count (a) *Acute lymphatic leukæmia* Red cells, 3 millions per c mm or less. Hb 50% or less. CI 0.8 to 0.9. There may be anisocytosis with a few megaloblasts or normoblasts. Platelets, usually reduced. White cells, 10,000 to 100,000 per c mm. The majority of cells are non-granular. Polymorphonuclears 2%, lymphocytes—some with "smeared" nuclei and lymphoblasts 98%. The lymphocytes may be small or large. The oxidase reaction is negative as the cells are agranular. (b) *Acute myeloid leukæmia* Red cells as above, but often megalocytes and some megaloblasts are seen. White cells 20,000 to 200,000 per c mm. Polymorphonuclears 2 to 4%. Lymphocytes 3%. Myeloblasts and myelocytes 93%. The myeloblastic cells give the oxidase reaction as they are granular. (c) *Acute monocytic leukæmia* Red cells as above. White cells, 50,000 per c mm. Monocytes 87%. Polymorphonuclears 4%. Lymphocytes 9%. Later a few monoblasts may be found. In some cases the blood changes are aleukæmic in type. Sternal puncture shows the bone marrow very cellular, 70 to 99% of the cells being primitive white cells of either the myeloid or lymphatic series.

**Differential Diagnosis** The onset of the illness may suggest a feverish cold or influenza. Hæmorrhage may point to a lesion of the lungs, stomach or intestines. The enlarged glands, fever and palpable spleen or liver occur also in glandular fever. The continuous fever may arouse suspicions of infective endocarditis, tuberculosis, or enteric fever. The hæmorrhages in the skin or mucous membranes might be due to purpura or scurvy. The diagnosis is established by the blood count and examination of the bone marrow, attention should be paid to the changes also present in the red cells, which are a distinctive feature of acute leukæmia as opposed to glandular fever.

**Course and Complications** The course usually is rapid, but remissions may occur in the monocytic variety. Subacute cases occur characterised by the bone marrow findings of acute leukæmia, the formation of tumours in the mediastinum, abdomen or bones (see chloroma, p. 502) and in which there is little or no fever, hæmorrhages are unusual, and the fatal issue may be postponed for about six months.

Complications include the hæmorrhages into various sites and leukæmic retinitis.

**Prognosis.** The outlook is hopeless; death usually occurs in a few days to a few weeks from the onset. A cerebral hæmorrhage may cause rapid death.

**Treatment.** This is symptomatic. Necrotic mouth lesions should be treated as described under agranulocytosis (see p. 507). X-ray treatment may cause a temporary disappearance or diminution of a mediastinal mass.

### Leukanæmia

#### (Erythroleucosis. Panmyelosis)

This condition may be an intermediate phase between pernicious anæmia and an anæmia of the leukæmic type. It is an acute and progressive disease, with symptoms resembling those of acute leukæmia. The blood count: Red cells, 3 millions per c.mm. Hb. 64%. C.I. 1.1. Anisocytosis, megalocytosis and a few megaloblasts may be seen. White cells, 26,000 per c.mm. Polymorphonuclears 60%. Lymphocytes 21%. Large mononuclears 9%. Myelocytes 10%. An occasional myeloblast may occur. The special features of these cases are that in the early stages they often resemble pernicious anæmia, both in the blood count with a high colour index and megalocytosis, and in the presence of achylia gastrica. They do not respond to liver treatment, and later show evidence of acute myelosis or acute lymphadenosis as judged by the occurrence of myeloblasts and myelocytes, or of lymphoblasts in the blood.

### Chloroma

#### (Chloroleukæmia)

**Definition.** A variety of subacute myeloid or rarely lymphatic leukæmia accompanied by the formation of tumours in the subperiosteal tissues and elsewhere.

**Etiology.** The cause is unknown. Chloroma occurs usually in male children.

**Pathology.** Greenish nodules of lymphoid tissue are found in all the organs of the body, except the brain, and they occur under the periosteum, and in the bone marrow, especially in the orbit, skull, long bones, vertebrae, ribs and sternum. The green colour, the cause of which is unknown, fades on exposure to air.

**Clinical Findings.** The symptoms may resemble those of acute leukæmia. Early nervous symptoms, such as pains in the back and legs and inability to walk, may be the first indication of ill health. In addition, an orbital tumour may cause protrusion of the eye, and swellings in the skull may result in facial paralysis, deafness or blindness. A sternal swelling may be apparent. The blood count resembles that of acute myeloid or rarely lymphatic leukæmia.

**Prognosis.** Death occurs rapidly, usually in 4 to 8 months from diagnosis.

**Treatment.** There is no cure and treatment is only symptomatic.

### Chronic Lymphatic Leukæmia (*Lymphadenosis*)

**Pathology** The lymph glands are enlarged, and on section show an excess of lymphoid cells. The spleen is enlarged, with lymphatic infiltration. The liver is enlarged, the periportal connective tissue is infiltrated with lymphocytes. The bone marrow of the long bones is grey and shows lymphoid metaplasia.

**Clinical Findings** The patient is usually a male of middle age. He complains of weakness, pruritus, bone pains, swelling of the tonsils, enlargement of the glands in the neck or elsewhere or of swelling of the abdomen, caused by the spleen.

**On Examination** Groups of enlarged glands may be seen and felt in the neck, axillæ, elbows and groins. They are soft early in the disease but later are hard, they are not tender. The skin is freely movable over them. In some instances there is a generalised cutaneous lesion resembling exfoliative dermatitis and small nodules may be felt in the skin (leukæmia cutis). The skin in certain areas may be slightly red and thickened owing to lymphatic hyperplasia. The tonsils may be enlarged owing to lymphatic infiltration. The spleen and liver are usually enlarged, often to a considerable degree. There is frequently a slight but irregular fever. Cranial nerve palsy has been noted in some cases, the VI and VII nerves being especially liable to be affected. The blood count. Red cells 3 millions per c.mm. or less. Hb 60% or less. CI 0.6. White cells about 90 000 per c.mm. Polymorphonuclears 2 to 8%. Small lymphocytes 92%. Large lymphocytes 2%. An occasional lymphoblast. The bone marrow obtained by sternal puncture shows 40 to 90% of the cells to be lymphocytes. The basal metabolic rate is raised.

**Differential Diagnosis** The disease must be differentiated from other causes of chronic glandular and splenic enlargement such as myeloid leukæmia, Hodgkin's disease, tuberculosis, syphilis, etc. Enlargement of the mediastinal glands may produce cough simulating whooping cough. The blood count establishes the diagnosis. There is, however, a variety of chronic leukæmia in which the total number of white cells is not increased but the differential count is similar to that given above. This is called *aleukæmic lymphæmia*. Examination of the bone marrow is of great help in establishing the diagnosis.

**Course and Complications** The disease usually pursues a chronic course of several years' duration. The number of large lymphocytes and lymphoblasts in the blood often increases, indicating the probability of an acute termination. The presence of myelocytes may be indicative of bone marrow stimulation. Complications include Venous thrombosis and rarely hæmorrhages as in the middle ear, retina or stomach.

**Prognosis** Death usually occurs in from 3 to 5 years from the date of diagnosis.

**Treatment** In addition to general hygienic measures the most hopeful line of treatment consists in the exposure of the spleen, chest and enlarged glands to X rays or radium. In order to prevent X ray

sickness, 2 mls of liver extract, such as Hepatex I.M., should be injected intramuscularly daily, during the course of X-ray treatment. Two ounces of dextrose in orangeade should be taken daily. A course of iron should also be given during the X-ray treatment, the following mixture being prescribed: Ferri et ammon. cit. gr. 30, glycerin. m. 15, aq. chlorof. ad fl. oz. 1. Fl. oz. 1 t.i.d. p.c. Arsenic should be given between the courses of X-ray treatment. This may be administered as intramuscular injections of sodium cacodylate gr. 1 for 20 injections, or liq. arsenicalis by mouth in doses increasing daily by m. 1, from m. 1 to m. 20 t.d.s. and then decreasing again to m. 1.

### Chronic Myeloid Leukæmia

(*Spleno-medullary Leukæmia. Myelosis*)

**Pathology.** The clotted blood may have a greenish-white colour. The lymph glands: These are only slightly affected. The mesenteric glands may be enlarged. The spleen is much enlarged and may weigh as much as 18 lbs. (normal weight is 5 to 6 oz.). There is perisplenitis. It is firm on section and whitish nodules are studded in its texture. There is myeloid metaplasia of the spleen substance and atrophy of the Malpighian corpuscles. The liver is enlarged, and areas resembling multiple abscesses are seen, consisting of myeloid metaplasia around the intralobular capillaries. Portions of the ileum may be infiltrated with myeloblasts resulting in ulceration and perforation. The bone marrow: This is greyish-red, and shows proliferation especially of myeloblasts. The kidneys show leukæmic infiltration.

**Clinical Findings.** The patient is usually a male between the ages of 20 and 45 years. The onset is insidious with weakness, pallor and dyspnoea, pain in the region of the spleen, abdominal swelling, or rarely priapism due to venous thrombosis may be the first symptom noted.

**On Examination:** The patient is pale. The spleen is enlarged, at times only just palpable, but in other cases filling nearly half the abdominal cavity, and friction may be heard over it. The liver is enlarged and palpable. Usually the lymph glands cannot be felt. There may be ascites or œdema of the legs. Occasionally small subcutaneous nodules of myeloid tissue are present, and in the later stages there are petechial or purpuric lesions of the skin. The urine often contains excess of uric acid, owing to the breakdown of leucocytes. It may be found that the temperature is slightly raised. The basal metabolic rate is raised. The blood: The uric acid may be increased. The blood count: Red cells, 3 to 5 millions per c.mm. or less. Hb. 50% or less. C.I. 0.7 to 1. The red cells may show anisocytosis, polychromasia, and a few normoblasts or megaloblasts. The platelets are usually first increased and later diminished. The white cells, 300,000 to 500,000 per c.mm. Differential count: Polymorphonuclears 40%. Eosinophils 5%. Basophils 2%. Small lymphocytes 10%. Large lymphocytes 5%. Large mononuclears 1%. Myelocytes 31% (usually neutrophil, a few eosinophil and basophil). Premyelocytes and myeloblasts 3%. In *eosinophilic leukæmia*, the leucocytes are chiefly mature eosinophil cells, with larger granules than are present



in normal eosinophils usually eosinophil myelocytes up to 2% are present. The bone marrow obtained by sternal puncture shows an increase in myeloblasts, promyelocytes and myelocytes. *Aleukæmic leukaemia*. This resembles aleukæmic lymphæmia mentioned on p. 503 but in this condition the abnormal white cells are of the granular type (myeloblasts and myelocytes) although the total white cell count is not increased. The diagnosis can be established by examination of the bone marrow.

**Differential Diagnosis.** Myeloid leukaemia must be differentiated from other causes of anæmia especially pernicious anæmia and from other conditions associated with enlargement of the spleen. Examination of the blood establishes the diagnosis.

**Course and Complications.** The course is usually prolonged for 4 or 5 years or more. Crises may occur, in which the patient becomes more ill with an increase in the myeloblasts. Towards the end the disease may pursue a very rapid course. Remissions do not occur spontaneously but may result from secondary infections or from treatment. Complications include venous thrombosis, hæmorrhages in internal organs and muscles, labyrinthine involvement causing deafness and vertigo and leukaemic retinitis.

**Prognosis.** The disease is fatal.

**Treatment.** The patient should be in bed taking an ordinary mixed dietary. X rays should be applied to the spleen or to the long bones, the spine and thoracic bones, liver extract, dextrose and iron being administered as described on p. 504. Alternatively radium may be applied over the spleen. Arsenic should be given as in lymphatic leukaemia, between the courses of X ray treatment. If this fails benzol (benzene) may be given by mouth in capsules containing benzol and olive oil m 5 of each, beginning with one capsule t d s, and increasing to 4 capsules t d s. The effect must be judged by blood counts as in some cases there is a rapid reduction in all the blood cells. The drug should be discontinued when the white cells are reduced to 20 000 per c mm. Splenectomy should not be performed.

### Agranulocytosis

(*Agranulocytic angina* *Granulocytopenia* *Malignant neutropenia*)

**Definition.** A disease characterised by a marked diminution in the number of granulocytes in the blood (neutropenia) with ulceration in the mouth, rectum or vagina.

**Etiology.** The cause is unknown. Two types are described.  
1 **Primary.** These cases are sometimes due to the use of amidopyrine (Pyramidon), Amidophen, Gardan or to barbiturates containing amidopyrine such as Allonal, Cibalgin, Compral, Somnosol, Veramon and Veropyron. Dinitrophenol used for slimming, benzol, bismuth and gold salts and the sulphonamides may also cause agranulocytosis. The patient may be sensitive to very small doses of amidopyrine.  
2 **Secondary** to some infection such as pneumococcal tonsillitis, pneumonia, osteomyelitis, sinusitis, staphylococcal septicæmia and liver

abscess. *Predisposing causes:* 1. Age: Usually adults. 2. Sex: Females predominate.

*Pathology.* There is a marked diminution or absence of myeloid cells (aplastic type) or of granulocytes (maturation type) in the bone marrow. No leucocidal substance is present in the blood.

*Clinical Findings.* The disease may have an acute onset with sore throat, dysphagia, headache, shivering, generalised myalgic pains and fever. In other cases the disease begins insidiously during gold or sulphonamide treatment. Chronic or recurrent cases also occur in which the white cells from time to time fall below 4,000 per c.mm., owing to a reduction of granulocytes, with accompanying symptoms of ill-health and a liability to septic infections.

*On Examination:* The temperature is raised to about 102° F. Ulceration is seen in the mouth, in such sites as the tonsils, pharynx or tongue. The cervical glands are in some cases enlarged and the spleen may be just palpable. There may be jaundice and necrotic lesions in the skin, rectum or vagina. There is no tendency to bleeding from mucous membranes. Meningitic symptoms may occur, the cerebrospinal fluid showing a paretic type of Lange reaction (see p. 290). The urine: A trace of albumin is usually present. The blood: There is a leucopenia, due to the marked diminution of the granulocytes. A typical count is as follows: White cells, 1,000 per c.mm. Polymorphonuclears 4%, mononuclears 16%, lymphocytes 80%. No immature white cells are seen. The red cell and platelet count are normal, or there may be a slight anæmia.

*Differential Diagnosis.* The throat lesion may suggest Vincent's angina or diphtheria. These are excluded by examination of the throat swabs and by the blood count. The leucopenia with relative lymphocytosis is differentiated from aleukæmic lymphæmia (see p. 503) by the absence of immature white cells. Monocytic angina somewhat resembles agranulocytosis. In the former the white cells show a high proportion of monocytes (up to 80%). In aplastic anæmia the red cell count is low.

*Course and Complications.* In acute untreated cases the course is usually rapidly progressive to a fatal issue. Complications include bronchopneumonia and jaundice.

*Prognosis.* Published results show a recovery rate of 74% of cases treated by Pentnucleotide. The response of the maturation type is more favourable than that of the aplastic type.

*Treatment. Prophylactic.* Amidopyrine is a dangerous drug and should only be taken under medical supervision. Gold salts should not be administered unless weekly leucocyte counts are made. Sulphonamides should not be given for longer than 7 to 10 days and drug fever is an indication for their immediate discontinuance.

*Curative.* No definite curative treatment is known. Good results have been obtained with the use of Pentnucleotide, 0.7 G. in 10 mls ampoules. The usual dose is 0.7 G. intramuscularly morning and evening until the white cell count begins to rise (usually the 4th or 5th day), and then one injection daily until the white cell count is normal.

In very acute cases 0.7 G. of Pentnucleotide in 100 mls of normal saline is injected slowly intravenously every morning for 4 days and 0.7 G. is injected intramuscularly in the evening. The intravenous injection should not be given if there is myocardial weakness. Blood transfusion is of doubtful value, but a small transfusion (500 mls) should be given if the patient is intolerant of Pentnucleotide, as shown by symptoms of cardiac distress after a trial dose. The local necrotic areas should be appropriately treated. Thus the mouth should be sprayed before feeding with Percaine 1% in glycerin 75% and water 24%, if pain prevents swallowing. The mouth should be swabbed after feeds with hydrogen peroxide (10 vols) diluted with an equal quantity of water, followed by a swabbing with a citric acid solution of gr 10 to 1 oz.

### Hodgkin's Disease

#### (*Lymphadenoma Lymphogranuloma*)

**Definition** A fatal disease characterised by enlargement of lymph glands with the formation of lymphogranulomatous tissue in the spleen and elsewhere.

**Etiology** There are various theories. Hodgkin's disease has been considered to be 1. An atypical form of tuberculosis. 2. A specific infective granuloma. 3. A neoplastic disease. 4. A reticulo-endotheliosis. 5. A virus infection.

1. Undoubtedly tuberculosis may supervene upon Hodgkin's disease but in the latter the primary changes are not tuberculous. There is no evidence that avian tuberculosis is the cause.

2. Spirochaetes, protozoa and diphtheroid bacilli have been incriminated as the causative organism without adequate proof.

3. The property of lymphadenomatous tissue to infiltrate muscle and bone suggests that it is allied to a sarcoma or endothelioma.

4. Overactivity of reticulo-endothelial cells leads to the formation of excess of lymphocytes and endothelial cells. The latter probably develop into giant cells.

5. The work of Gordon suggests the probability of a virus infection, the causal agent being 'very minute spherical or oval elementary bodies (E. B.'s)' found in the affected lymph glands during the acute stage (see p. 509).

**Pathology** The lymphatic glands and spleen are chiefly involved, but in some cases no enlargement of these structures can be seen at autopsy and the diagnosis can only be established by finding lymphadenomatous tissue in the liver and other organs on microscopical examination. The glands are enlarged in different parts of the body, such as the neck, mediastinum, axillæ, abdomen and groins. They are firm and greyish white on section, although fatty degeneration and tuberculous caseation may occur. Microscopically the fibrous tissue is increased; there is an infiltration of lymphocytes and peculiar lymphadenoma or giant cells (Dorothy Reed cells) with central nuclei are present. The endothelial or epithelioid cells are increased, and often there are masses of eosinophil cells. The spleen. This is made

ately enlarged and firm. On section there are white areas of lymphogranulomatous tissue ("hard-bake" spleen). The liver may also be enlarged and contain lymphogranulomatous tissue. The kidneys may be similarly affected, and very rarely the bladder is involved. The lungs may be studded with small nodules. The vertebral canal: Lymphogranulomatous tissue may spread from the periosteum into the vertebral canal and compress the cord or spinal nerve roots. The bodies of the vertebræ may be involved. The stomach and intestines may show hyperplasia of lymphatic tissue. In some cases the disease appears to start in the reticulo-endothelial cells of the thymus. The bone marrow may be infiltrated with lymphogranulomatous tissue.

**Clinical Findings.** The patient is usually a male between 15 and 45 years of age. Females are affected only half as frequently. The onset is generally insidious, the first symptom being usually enlargement of glands in the neck or axilla. In other cases weakness or dyspnoea may first attract attention. Further symptoms depend upon the site of the enlarged glands or of the lymphogranulomatous infiltration. Thus pain may result from pressure on nerve trunks going to the extremities or chest, intense dyspnoea may be caused by tracheal or bronchial constriction, there may be cutaneous irritation, or diarrhoea from intestinal involvement. Frequency of micturition and dysuria are symptoms of bladder involvement. In latent cases a persistent irregular or intermittent fever may be an early symptom.

**On Examination:** There is usually pallor, and the enlarged glands may be seen projecting from the side of the neck or forming a collar. The glands may also be visible or palpable in the axillæ or groins. The glands are firm, slightly mobile and the skin over them is neither adherent nor discoloured. Examination of the chest may reveal signs of mediastinal glandular enlargement, such as D'Espine's sign (see p. 198) or weak air entry over the apex of one lung, unequal pupils or a pleural effusion. Dilated venous radicles may be seen over the upper part of the chest, back or front. The spleen is usually moderately enlarged, the liver just palpable, and ascites may be present, or jaundice due to pressure of glands in the portal fissure. Enlarged abdominal glands cannot usually be felt. Pressure on the cord or spinal roots may result in sensory changes in the extremities, or some degree of weakness or paraplegia. The skin is usually pale, but pigmentation may be seen apart from that resulting from the therapeutic use of arsenic, especially if the abdominal glands are involved. Other cutaneous lesions include erythematæ, generalised exfoliative dermatitis, and rarely small nodules of lymphogranulomatous tissue may be felt under the skin. Herpes zoster is usually associated with arsenical treatment, but it may occur apart from this and be generalised.

**The Relapsing Type (Pel-Ebstein Syndrome).** As mentioned above, there is usually slight irregularity of temperature in all cases of Hodgkin's disease, but periodical waves may occur with apyrexial intervals. The pyrexial periods usually last for 5 or 6 days and recur at intervals of 15 to 36 days, the span, which is the distance between the crests of the successive waves, being fairly constant in each case. During

the fever the patient feels more ill, may vomit and the spleen and glands often enlarge. In the intervals the patient feels comparatively well and may gain weight. The blood. There is no characteristic change. In some cases there is a leucocytosis of 20,000 per c mm. or more, even in the afebrile periods, with an excess of polymorphonuclears, reduction of lymphocytes and slight excess of monocytes. In other cases there is leucopenia. In acute cases the number of eosinophils may be much increased, and they have large granules. When the bone marrow is involved myelocytes and myeloblasts appear in the circulation. The red cell count may show a chronic hæmolytic anaemia, but in some cases a macrocytic anaemia has been described, closely resembling pernicious anaemia, but with a normal or increased fragility of the red cells.

**Differential Diagnosis.** This involves a consideration of other causes of glandular and splenic enlargement, of mediastinal pressure, of ascites and jaundice, and of irregular or periodical pyrexia. Enlarged glands may result from tuberculosis, leukaemia, lymphosarcoma, syphilis, sepsis and glandular fever. Mediastinal pressure may be due to a new growth, aneurysm or enlarged glands from several causes. Unexplained pyrexia may be due to tuberculosis, infective endocarditis, enterica group infections, brucella infections, malaria etc. In all cases the blood must be examined, and if possible a superficial gland should be removed and examined microscopically. Often, however, the pathological report is indefinite. Gordon's biological test is of some value, but the encephalitogenic agent appears to be present in eosinophil cells. The positive test therefore depends upon the presence of eosinophils in the glands. A broth emulsion of a gland is injected intracerebrally into a rabbit. If the test is positive the animal develops spastic paralysis of the hind limbs, and ataxia with inco-ordination and death may occur. The test is positive in about 70% of cases of Hodgkin's disease.

**Course and Complications.** Certain types are described according to the course pursued. 1 Chronic Hodgkin's disease. Here the disease is progressive but interrupted by remissions. Life may be prolonged to four or five years. 2 The relapsing type. Described above. 3 The acute type (Hodgkin's sarcoma), in which the disease is rapidly fatal. Complications result from mediastinal pressure, intercurrent infection, amyloid degeneration or tuberculous infection.

**Prognosis.** The disease is invariably fatal.

**Treatment.** There is no known cure. Adequate rest, fresh air and nourishing food should be ensured. Arsenic usually causes a temporary improvement. It is best given in courses of intramuscular injections of sod. cacodylat gr 1 daily for 12 doses, with a rest on the seventh day. After an interval of 3 weeks a second course can be given. X ray treatment or radium exposures to the affected areas of the body also cause temporary improvement, especially if combined with weekly intravenous injections of 0.3 G. neoursphenamine. During the X ray treatment liver extract and dextrose should be administered as described on p. 504.

## Splenic Anæmia

*(Banti's Disease)*

**Definition.** A disease characterised by anæmia, enlargement of the spleen, cirrhosis of the liver and gastro-intestinal hæmorrhages. Some authorities differentiate splenic anæmia from Banti's disease, maintaining that cirrhosis of the liver does not occur in the former; others deny the concept that splenic anæmia or Banti's disease is a primary disease of the spleen. They believe that the condition is one of congestive splenomegaly and that the pathological findings are always due to mechanical obstruction of blood flow in the portal system, the commonest cause being hepatic cirrhosis.

**Etiology.** The cause is unknown. Splenic anæmia is often believed to be a primary disease of the spleen. It may be an infection due either to a streptothrix or to the *Bacterium commune* (*B. coli*). It may be secondary to an infective thrombo-phlebitis of the splenic or portal veins.

**Pathology.** The spleen is enlarged and smooth. It is firm on section owing to thickening of the capsule and trabeculae. Fibrosis occurs around the central artery of the Malpighian bodies. The lining cells of the sinuses of the pulp are hypertrophied and infarcts may be present. The liver may be slightly enlarged, or small and cirrhotic. The splenic veins are seen to be tortuous and dilated, at splenectomy. Phlebitis is seen in some cases in the splenic and portal veins, and the œsophageal and gastric tributaries are dilated.

**Clinical Findings.** The patient is usually a young adult male. The disease is rare in children and after middle age. The onset is insidious. The first symptoms of ill health may be lassitude, pallor, epistaxis, hæmatemesis, melæna, or enlargement of the abdomen. Banti's disease is described as passing through three stages:—*The first stage*: There is anæmia, and enlargement of the spleen, but no jaundice. The spleen may weigh as much as 25 oz., and yet not be palpable. The blood: There is a hæmolytic anæmia. No nucleated red cells are present. The white cells often show a leucopenia with a relative lymphocytosis. The fragility of the red cells is normal. The blood platelets may be increased or diminished, constituting a thrombocythæmic or a thrombocytopenic variety of the disease. *The second stage*: After 2 or 3 years the liver enlarges and there is slight jaundice. *The third stage*: In another 2 or 3 years the liver shrinks and ascites appears. It must be understood that all cases do not pass through these stages. Severe hæmorrhage may occur from the œsophagus, stomach, intestines, kidneys or nose, and purpura may be seen. The lymphatic glands do not enlarge.

**Differential Diagnosis.** In establishing the diagnosis it is necessary to consider other causes of severe anæmia, of epistaxis and internal hæmorrhages, of tumours in the left hypochondrium, and of enlargement of the liver and spleen, or of ascites. The blood count demonstrates the anæmia. The tumour in the left hypochondrium presents the characteristic features of an enlarged spleen (see p. 518). A gastric

ulcer and carcinoma of the stomach or colon may present difficulties, but an opaque meal usually will exclude these lesions. In acholuric jaundice the fragility of the red cells is increased, and bleeding from mucous surfaces does not usually occur. The supporters of the view that Banti's disease is a separate entity state that in cirrhosis of the liver the spleen is not usually enlarged before the liver enlarges, and the splenic enlargement in cirrhosis is not usually as marked as in Banti's disease. With primary thrombosis of the portal or splenic vein there is usually leucocytosis and the abdominal symptoms are more acute.

**Course and Complications** The course is slowly progressive, as described above, although sudden death may occur from hæmorrhage. Intercurrent infections may ensue.

**Prognosis** This is very grave, if untreated the disease is almost always fatal.

**Treatment** Iron should be given in adequate doses, such as freshly prepared Bland's pill (pil ferri carbonatis B.P.), cut into two parts before being swallowed, gr 15, t i d, or ferri et ammon cit gr 30 t i d, for eight weeks. This may be combined with X ray treatment to the spleen. In hæmorrhage a drip transfusion may save the patient's life, although usually only temporarily, as recurrences are very liable to occur at intervals of months or years. Splenectomy is of doubtful value and the operation is not devoid of risk. In cases in which the platelet count is high, thrombosis of the mesenteric or portal vein may occur 8 to 10 days after the operation and cause death. Further severe hæmorrhages are liable to occur after splenectomy in at least 50% of patients who suffered from hæmorrhage before the operation.

## THE LIPOIDOSES

Under this heading are classified three diseases characterised by a disturbance of lipid metabolism.

### Gaucher's Disease

**Definition** A rare disease characterised by enlargement of the spleen, the presence of Gaucher cells in the spleen and other organs, and a tendency to a familial incidence.

**Etiology** The cause is unknown. It is regarded as an inborn error of lipid metabolism.

**Pathology** The spleen is enlarged and the capsule thickened. Infarcts may be seen on section. Microscopical examination reveals the typical Gaucher cells. They are enlarged reticulum cells, derived from the reticulo endothelial system containing a lipid, kerosin. Similar cells may be found in the liver, mesenteric glands and bone marrow, and rarely deposits in the lungs.

**Clinical Findings** The onset is in childhood and females of the Jewish race are especially affected. The patient may complain of abdominal swelling or discomfort, of pains in the muscles and bones, and of a tendency to bleeding from the nose or gums.

**On Examination:** The enlarged spleen is usually found on routine examination. The liver may be just palpable or considerably enlarged. There is often pigmentation of exposed areas of the skin, and there may be slight jaundice. A brown-yellow fatty thickening (pinguecula) may be seen on the conjunctivæ. The blood: There may be some degree of hypochromic anæmia and leucopenia. The blood platelets are slightly diminished. Examination of the bone marrow obtained by sternal puncture shows typical Gaucher cells.

**Differential Diagnosis.** The diagnosis can only be established with certainty during life by splenic or sternal puncture, and the discovery of Gaucher cells in the material removed. The familial incidence, age of onset, enlargement of the spleen, chronic course and the usual maintenance of fair health suggest the diagnosis.

**Course and Complications.** The course is prolonged, and the patient usually survives for many years. In the later stages the liver may be considerably enlarged.

**Prognosis.** The ultimate outlook is unfavourable.

**Treatment.** The only hope of cure is splenectomy. As the operation is not devoid of risk, it is doubtful whether it should be performed. Iron should be administered in adequate doses for the anæmia.

### Niemann-Pick Disease

This disease affects infants usually of the Jewish race. It is characterised by enlargement of the spleen, liver and superficial lymph glands. The lipoid cells are present in various organs of the body including the central nervous system. Phosphatide metabolism is disturbed. The skin is pigmented on exposed areas and the blood shows a hypochromic anæmia, with lipoid vacuoles in the white cells. The blood cholesterol is usually raised. Cells obtained by sternal puncture show a typical foam appearance. The disease appears to be related to the Tay-Sachs syndrome (see p. 829). It is usually fatal before the age of two years and no cure is known.

### Hand-Schüller-Christian Disease

#### (Xanthomatosis of Bones)

This is another disorder of lipoid metabolism (cholesterol esters), which usually affects males. Accumulations of lipoid material with formation of granulation tissue or fibrotic areas containing cholesterol crystals occur in bones, especially in the membrane bones of the skull. The spleen, liver, kidneys and lymph glands are often enlarged. The blood cholesterol may be raised except in periods of remission. The disease shows itself in early childhood and is characterised by bony softening, exophthalmos and diabetes insipidus. Granulation tissue involving the orbit results in exophthalmos and when it invades the sella turcica diabetes insipidus results. Rarely the disease first shows itself in adult life, the bony changes being chiefly in the long bones, and exophthalmos is not necessarily present.



### Enlargement of the Spleen

The causes of enlargement of the spleen may be classified as follows

1 *Protozoal infections and parasitic worms* such as malaria, kala azar and schistosomiasis 2 *Bacterial infections*, such as enterica group organisms streptococci in septicaemia etc 3 *Hæmopoietic diseases*, such as pernicious anaemia, leukaemia splenic anaemia von Jaksch's anaemia Hodgkin's disease, Gaucher's splenomegaly, acholuric jaundice polycythæmia rubra etc 4 *Specific infective granulomata*, as in tuberculosis and syphilis 5 *Deficiency diseases* such as rickets 6 *Vascular disturbances* such as infarction passive hyperæmia from torsion of the pedicle thrombosis of the splenic vein cirrhosis hepatis, pressure of enlarged glands on the portal vein and very rarely congestion due to heart failure 7 *Cysts* such as a hydatid and dermoid, and a serous or hæmorrhagic cyst 8 *Tumours* such as primary or secondary sarcoma or secondary carcinoma Simple tumours are rare, such as a fibroma and hæmangioma 9 *Abscess* such as a septic infarct 10 *De generation*, as in amyloid disease

The signs of enlargement of the spleen are as follows A tumour may be seen or felt in the left hypochondrium The spleen enlarges downwards and forwards towards the umbilicus, and may extend also downwards towards the anterior superior spine of the left ilium and to the right of the mid line A notch can usually be felt on its anterior border The loin is not filled by the tumour It moves slightly with respiration, unless fixed by adhesions It is dull on percussion and the dulness extends backwards to fuse with the normal area of splenic dulness over the left lower chest behind There is no band of intestinal resonance running across the dull area but the flank behind the tumour is usually resonant In some cases the spleen is enlarged, but not palpable (see p 510)

**Differential Diagnosis** Other swellings in the left hypochondrium which must be excluded are a renal or suprarenal tumour, a growth of the stomach or colon a pancreatic or ovarian tumour, and tuberculous peritonitis With renal tumours there are generally urinary changes, and the characteristic signs are present (see p 480) An opaque meal or enema will assist in excluding a growth in the stomach or colon Pancreatic tumours, when palpable, are usually cysts appearing on the surface near the midline An ovarian tumour could only be mistaken for a very large splenic tumour, but pelvic examination will indicate its origin Tuberculous glands with peritonitis do not usually give the typical signs of an enlarged spleen

### Rupture of the Spleen

This is a rare event It may follow a blow over the spleen or a crushing accident The spleen may rupture spontaneously as a complication of malaria infarction typhoid fever, pregnancy or nitro benzene poisoning Clinically the patient suffers from severe pain and shock with the signs of internal hæmorrhage and a laparotomy is necessary to save life

### Movable Spleen

This is rarely met with in generalised visceroptosis. If it gives rise to dragging and discomfort it may be supported by a pad and belt.

### Perisplenitis

This occurs especially in connection with splenic infarction. The patient complains of pain over the spleen, increased on respiration. A coarse rub may be heard over the spleen, at the back of the chest or in the left hypochondrium.

### Erythrocytosis

**Definition.** An increase in the number of red cells in the blood, due to some known stimulus.

**Etiology.** The bone marrow is provoked to increase its output of red cells under various conditions interfering with the oxygen supply, such as high altitudes, congenital heart disease, pulmonary arteriosclerosis (*Ayerza's disease*), emphysema, heart failure, *Cushing's syndrome*, etc.

### Erythræmia

(*Polycythæmia Rubra or Vera. Vaquez's Disease. Osler's Disease*)

**Definition.** A condition characterised by an increase in the number of red cells in the blood, with splenomegaly.

**Etiology.** The cause is unknown. It has been suggested that there is thickening of the vessels supplying the bone marrow, the resultant anoxia stimulating erythropoiesis.

**Pathology.** There is hyperplasia of marrow in the long bones. The spleen is enlarged and may contain infarcts. Tuberculosis of the spleen has been found in some cases.

**Clinical Findings.** The patient is usually middle-aged, of either sex; other members of the same family may be similarly affected. The onset is insidious with headache, lassitude, giddiness, dyspnoea and insomnia. Epistaxis, or a hæmorrhage from the stomach, uterus and rarely from the lungs may occur. The patient may notice that his face and hands are becoming high coloured.

**On Examination:** The face is typically brick-red, and the ears, lips, hands and fingers may be cyanosed. The spleen is enlarged to a variable extent. The heart is not usually enlarged. The blood: The total volume is two to three times the normal. Red cells, 7 to 12 millions per c.mm. Hb. 125 to 150%. C.L. 0.8 to 0.9. There is polychromasia, and a few normoblasts and reticulocytes are present. The platelets may be increased. White cells, 15,000 to 20,000 per c.mm. Polymorphonuclears 70 to 90%. A few myelocytes are present. The urine: There is often a trace of albumin and a few casts may be present. Acholuric jaundice may be present. The blood pressure may be raised, as in polycythæmia hypertonica of Gaisböck (see p. 266). In these cases there is usually no splenomegaly.

**Differential Diagnosis.** Erythræmia must be distinguished from

erythrocytosis, for which some causative factor can be found. Enterogenous cyanosis is recognised by spectroscopic examination.

**Course and Complications** The disease pursues a chronic course, which is interrupted by remissions. In some cases the red cell count may drop to 3 millions, with an increase of immature white cells, resulting in a myeloid leukaemia or an aplastic anaemia before death occurs. Complications include cerebral thrombosis and heart failure.

**Prognosis** Death usually takes place in the space of 3 to 8 years from diagnosis, but the phenylhydrazine treatment has improved the prognosis, the blood count remaining normal for several years.

**Treatment** The patient should not eat any meat, liver, kidneys, meat soups or eggs in order to reduce the iron content of the diet. Vene section affords temporary improvement, 500 mls should be removed twice a week until the Hb falls to about 80%, red cells to about 5 millions and the hæmatocrit reading to about 45%. Alternatively phenylhydrazine hydrochloride may be cautiously administered, provided there is no evidence of disease of the liver or kidneys. It is given by mouth in a cachet in doses of 0.1 G daily for 10 days. The effect must be judged by blood examinations. An increase in the number of reticulocytes or in the number of immature white cells is an indication for its discontinuance. Phenylhydrazine has a cumulative effect and may produce aplastic anaemia. If it is well tolerated a second course may be given after a fortnight's interval. Acetylphenylhydrazine is said to be less toxic, 0.1 G is given 2 or 3 days a week for several weeks. Splenectomy is not advisable. Some improvement has been obtained after X-ray treatment of the long bones, if care is taken to avoid stimulation of the bone marrow or the production of aplastic anaemia.

### Enterogenous Cyanosis

(*Sulphæmoglobinæmia Methæmoglobinæmia*)

**Definition** Cyanosis, due to the presence of sulphæmoglobin or methæmoglobin in the blood.

**Etiology** Stockvis, in 1902, described what he called enterogenous cyanosis, in a patient suffering from chronic dysentery whose blood contained methæmoglobin. In these cases it is thought that nitrites are absorbed from the intestines into the blood, and these reduce the hæmoglobin. The reduced hæmoglobin combines with the small amount of sulphuretted hydrogen normally present and sulph. Hb or met Hb is formed. A nitrifying organism (*nitroso-bacillus*) has been isolated from the saliva and faeces in some cases, and reducing substances are present in the serum, urine and saliva of patients suffering from the disease. In 1925 Snapper suggested that the hæmoglobin is sensitised by aniline drugs and then unites with sulphides absorbed from the intestines. Potassium chlorate, Bromo-seltzer and drugs of the coal tar group, such as sulphonal, Trional, and Antihæmia, may cause methæmoglobinæmia. Methæmoglobinæmia has also been met with in rats associated with an infection with the *Bacterium enteritidis* (Gaeriker), and the *Bacterium commune* (*B. coli*) has been isolated from the blood in one case in man. Sulphæmoglobinæmia is not

uncommon in connection with sulphonamide therapy. Whether sulphæmoglobinæmia or methæmoglobinæmia develops in sensitised patients appears to depend upon the degree of constipation present.

**Clinical Findings.** The patient is usually an adult of either sex. The onset of symptoms is insidious with headache, weakness, fainting attacks and nervous instability. Nausea, vomiting and abdominal pains may be complained of. There is mild or transitory constipation with methæmoglobinæmia and severe and chronic constipation with sulphæmoglobinæmia.

**On Examination:** The patient has a peculiar cyanosis, which is mauve-lavender in colour in sulphæmoglobinæmia and blue-chocolate-brown in methæmoglobinæmia. No physical cause for the cyanosis can be found on clinical examination. The blood: Spectroscopic examination shows the band of met. Hb. or sulph. Hb. between the C and D lines of the spectrum, further spectroscopic tests differentiating them. The pigment absorption band may be seen with a hand spectroscope through the lobe of the ear, but for accurate determinations laked diluted blood should be examined in the laboratory and special differentiating tests applied. The pigment is not present in the serum or in the urine.

**Differential Diagnosis.** Other causes of cyanosis, especially diseases of the heart and lungs, are excluded on clinical examination.

**Course and Complications.** The course in the enterogenous cases is prolonged, and complete recovery is doubtful. In some cases intermissions and relapses correspond with periods of freedom from, or affection with marked constipation.

**Prognosis.** The condition is not fatal, but a permanent cure is unusual.

**Treatment.** In all cases an exhaustive search should be made to exclude the use of such sensitising drugs as phenacetin, antifebrin (acetanilide), Trional (methylsulphonal B.P.), sulphonal and potassium chlorate. The constipation must be treated by the regular use of laxatives, avoiding those containing sulphur, and by washing out the intestine. For this purpose the patient may swallow a duodenal tube daily, and the whole of the intestine is then washed out with two pints of normal saline. The diet should be rich in fats, and ammon. chlorid. gr 15 should be given by mouth four times a day, in the form of two 0.5 G. stearetttes. A mixture containing 5% CO<sub>2</sub> in oxygen should be inhaled from a gas mask three times a day until the respiration rate is raised to 30 a minute. As the cyanosis diminishes the duration of each inhalation is reduced. The cyanosis can often be abolished by the administration of a methylene blue pill, gr. 2, 3 or 4 times a day. If the nitroso-bacillus is found in the mouth or faeces a vaccine should be prepared and administered, in doses of 10 millions increased to 500 millions.

## PURPURA

**Definition.** A condition characterised by extravasation of blood into the skin and mucous membranes, due to capillary hæmorrhage.

**Etiology** Purpura is a symptom occurring in many pathological states. For clinical purposes it is divided into a primary and secondary group. Primary or idiopathic purpura is also known as the *hæmorrhagic diathesis*, it arises apart from any known cause. Secondary or symptomatic purpura is a symptom of some recognisable disease. The following varieties are thus described.

• 1 *Primary or Idiopathic Purpura* The cases fall into two groups (a) hæmorrhagic purpura including purpura simplex, purpura hæmorrhagica and purpura fulminans and (b) anaphylactoid purpura which includes purpura rheumatica and Henoch's purpura.

2 *Secondary or Symptomatic Purpura* This may be associated with (a) Infections such as septicæmia and infective endocarditis (b) Fevers such as cerebro spinal fever, typhus fever, measles, small pox or scarlet fever (c) Hæmopoietic diseases such as leukaemia, pernicious anemia and Hodgkin's disease (d) Avitaminosis as in scurvy (vitamin C lack) and deficiency of vitamin P in the diet (e) Chemical causes such as iodides, mercury, quinine, copaiba, cubeb, bella donna, ergot, chloral hydrate, gold, arsenic, turpentine, phenacetin, Sedormid, salicylic acid and snake venom (f) Cachexia as in carcinoma, tuberculosis, old age and chronic nephritis (g) Nervous and endocrine disorders such as tabes dorsalis, transverse myelitis, neuralgia, fright and hysteria (h) Mechanical causes such as pressure on the skin, whooping cough, violent vomiting or epilepsy (i) Severe jaundice.

**Pathology** In hæmorrhagic purpura all the elements of the blood pass through the capillaries. In anaphylactoid purpura the serum alone may pass out into the tissues in some parts of the body, whereas the lesions in other sites are definitely purpuric. In purpura simplex there may be extravasation of red cells only. The lesions produced are thus variable. pinpoint spots constitute petechiæ; the purpuric spots are about 1 to 3 mm in diameter and larger extravasations several inches across are known as ecchymoses. The bleeding may occur into or under the skin, into or from mucous or serous membranes into the interstitial tissue of internal organs and into the eye. It must be clearly recognised that purpura does not depend merely upon deficiency of platelets in the blood, as there may be a complete absence of platelets without hæmorrhages. The platelets are much diminished in purpura hæmorrhagica (essential thrombopenia) and to a lesser degree in the other varieties of purpura depending upon the severity of the bleeding. The platelets are produced in bone marrow from megakaryocytes and destroyed by the spleen. They probably protect weak surfaces in the capillaries by forming a layer over the intima at such spots and thus help to prevent extravasation of blood. The essential lesion in purpura appears to be a temporary alteration in the permeability of the capillary endothelium. This may be due to toxins or to proteins to which the patient is sensitive, but this is uncertain. As long as the capillary endothelium is normal extravasation of blood does not occur even with very low platelet counts. The coagulation time of the blood is normal in purpura although the clot retracts

slowly; the bleeding time is much prolonged in primary purpuras when the platelet count is low. After splenectomy the platelet count rises very rapidly, due partly to an increased output from the marrow and partly to lack of destruction by the spleen, but it may return to its original low level although the patient is relieved of his symptoms.

### Purpura Simplex

**Clinical Findings.** The patient is usually a child or young adult, who gives a history of slight malaise, with perhaps headaches, fleeting pains in the joints or diarrhoea. Familial purpura simplex is described. Lack of vitamin P in the diet may result in the appearance of petechiae in the skin. The patient complains of lassitude with pains in the shoulders and legs.

**On Examination:** Small purpuric spots are seen chiefly on the extensor surface of the legs. The trunk, arms and rarely the face may be involved. The spots come out in crops and the temperature may be slightly raised. Hæmorrhages do not occur from the mucous membrane. The blood: The platelets are usually normal. The coagulation time is normal. The bleeding time is normal.

**Course and Complications.** 5 or 6 weeks may elapse before the spots finally disappear. Recurrences are often noted.

**Prognosis.** This is good.

**Treatment.** The patient should be in bed until the purpura disappears. A course of liq. arsenicalis is usually prescribed, but its value is very doubtful.

### Purpura Hæmorrhagica

(*Morbus Maculosus of Werlhof. Essential Thrombopenia. Purpura Thrombopenia. Thrombocytolytic Purpura. Thrombocytopenic Purpura.*)

**Definition.** A disease characterised by hæmorrhages into the skin, mucous membranes and internal organs, with a low platelet count.

**Etiology.** Two types are described: 1. *Idiopathic.* 2. *Secondary* to drugs such as Sedormid, iodides, gold, arsenic, quinine, etc., and possibly to sepsis.

**Pathogenesis.** The constant low blood platelet content may be due to: 1. A bone marrow maturation defect. This may occur in the *secondary variety of the disease due to drug idiosyncrasy.* 2. *Increased destruction of platelets by clasmocytes in the spleen (Kaznelson's view).* 3. *Increased capillary permeability, causing a loss of platelets.*

The bone marrow is normal in idiopathic cases, but may show decrease of megakaryocytes with involution forms in Sedormid purpura. The spleen is normal. After splenectomy the hæmorrhages often cease, although the platelet count, after a preliminary rise, may fall. The platelet deficiency therefore appears to be only one factor in the hæmorrhagic tendency.

**Clinical Findings.** The disease may show itself in an acute or chronic form. The patient is often about the age of puberty, but the purpura may occur at any age and more frequently in women. The

onset is often sudden with a hæmorrhage from the nose, stomach, kidneys or uterus

**On Examination** Purpuric spots and ecchymoses may be found on the body, the gums may bleed, but are not spongy as in scurvy. The temperature is often raised. The blood. The platelets are usually much reduced and may disappear altogether, although the initial symptoms may appear with a platelet count of 100 000 per c mm. The coagulation time and prothrombin time are normal. The bleeding time may be prolonged to several hours. The retraction of clot in shed blood is poor. There is usually a hypochromic anæmia and the reticulocytes may be increased up to 10% or more. A few nucleated red cells may be seen. There may be a leucocytosis or a leucopenia with relative lymphocytosis. Sternal puncture shows no abnormal cells and the megakaryocytes are normal in idiopathic cases.

**Differential Diagnosis** Hæmophilia can usually be excluded on clinical grounds. The platelet count and bleeding time are normal and the coagulation time is delayed. Hereditary purpura hæmorrhagica (constitutional hæmogenia) is a disease resembling purpura hæmorrhagica clinically, but differing in its tendency to hereditary transmission, its preference for the female sex, and the often fatal results of splenectomy. In only about 50% of cases is the platelet count low. In scurvy the hæmorrhages are usually in deeper tissues, such as the thighs the gums are spongy, and there is a history of avitaminosis. In acute leukaemia, the clinical picture may closely resemble that of the hæmorrhagic diathesis. The blood count usually serves to differentiate but there may be a leucopenia with relative lymphocytosis in acute leukaemia. The capillary resistance test of Hess may be of value in a doubtful case. The upper arm is compressed with the armlet of a sphygmomanometer, the pressure being maintained for 5 minutes mid way between the systolic and diastolic readings. In purpura hæmorrhagica purpuric rashes usually appear on the forearm. Some cases diagnosed as essential thrombopenia prove to be congenital nasal telangiectases.

**Course and Complications.** The course may be rapidly fatal or more prolonged with intermissions, or definitely chronic with recurrent hæmorrhages and purpura for several years. Cerebral or cerebro spinal hæmorrhage may occur as a complication.

**Prognosis** This is always very grave in acute cases. Chronic cases are more benign and there is a tendency to spontaneous improvement.

**Treatment** The various measures which should be tried to arrest the hæmorrhages include a blood transfusion of 300 mls every 3 to 5 days, the daily intravenous injection of 400 mg ascorbic acid dissolved in 8 mls of normal saline for 4 days, or the intravenous injection of 20 mls of a 1% solution of congo red. In chronic cases some good results have been obtained by the subcutaneous injection of 0.4 to 1 ml of 1/3 000 moccasin venom twice a week. If these fail a splenectomy should be performed in both acute and chronic idiopathic cases before the patient becomes too ill. If the patient recovers from the operation, relapse after splenectomy is rare, although it has occurred apart from there being any accessory splenic tissue in the body after the operation.

Splenectomy should never be performed if the patient has taken a drug which is likely to be the causative agent, or if he has any obvious source of infection, or if the bone marrow show diminution of megakaryocytes.

### Purpura Fulminans

A hyperacute variety of purpura which causes death in 1 to 5 days. Infants are chiefly affected, the temperature is raised, and large subcutaneous ecchymoses are seen. The blood shows an anæmia with a normal number of platelets.

### Schönlein's Purpura

(*Anaphylactoid Purpura. Allergic Purpura. Purpura Rheumatica. Peliosis Rheumatica*)

**Etiology.** The cause is unknown. There is no evidence that Schönlein's purpura is a rheumatic infection. It may be due to sensitivity to proteins or articles of food.

**Pathology.** At some sites in the body the capillaries allow transudation of plasma, and at others diapedesis of red cells occurs.

**Clinical Findings.** The patient is often a young adult male. There may be fever and sore throat at the onset, with pain in various muscles and joints. Swelling of joints may also be noted.

**On Examination:** The temperature may be raised to about 100° F. for the first few days of the illness. The affected joints, such as the knees or ankles, are slightly swollen and tender, but the skin is not discoloured. The skin: The cutaneous changes include purpuric spots, usually on the extensor surfaces of the legs, and urticarial wheals. Angio-neurotic swellings may appear in the legs, feet, face or hands. The blood: Usually the platelets are not diminished, and the bleeding and coagulation times are normal.

**Differential Diagnosis.** The fever, sore throat and joint pains are suggestive of acute rheumatism, and hæmaturia of acute nephritis. The purpuric spots and urticarial swellings are diagnostic, and there is no response to salicylates.

**Course and Complications.** The course may be prolonged for several weeks and relapses occur. In some cases there are intestinal symptoms characteristic of Henoch's purpura. The urine may contain albumin and blood.

**Treatment.** The patient must be kept in bed. Calcium lactate gr. 10 t.i.d.s. should be given. Later, any septic focus in the mouth or elsewhere should receive treatment. In cases associated with nutritional deficiency vitamin P should be administered in the form of Hesperidin tab., 0.25 G., four times a day.

### Henoch's Purpura

**Definition.** This is a more severe form of Schönlein's purpura, characterised by abdominal crises due to an exudation of plasma or blood into the wall of the intestines.



**Clinical Findings** The patient may be an infant or adult, who is seized with severe abdominal pain, vomiting, diarrhoea or constipation, and at times pains in the joints

**On Examination** The abdomen is rigid during the attack. There may be no purpura, or careful search may reveal the presence of a few spots. Urticaria and swelling of the joints may also be present. The blood. The platelets are usually normal and there is no change in the bleeding or coagulation times. Blood and mucus may be passed per rectum, and the temperature is raised.

**Differential Diagnosis** The disease is liable to be mistaken for an intussusception in an infant or for a mesenteric thrombosis or other variety of abdominal emergency in an adult. A very careful search should be made for purpuric spots, before any operation is performed.

**Course and Complications** An attack usually passes off in a day or so. Complications include hæmaturia, intussusception and cerebral hæmorrhage. Relapses are prone to occur.

**Prognosis** This is always serious.

**Treatment** The patient must be kept in bed, and an icebag applied locally to the abdomen to relieve pain. If this is not successful the chlorof et morphin co (B.P.C.) in 5 to 10 may be given to an adult and repeated if necessary. Vitamin P is worthy of trial administered as above. Antistreptococcus serum in doses of 25 mils may be given by mouth every other day for 3 doses or calcium lactate gr 10 t.d.s. by mouth.

### The Secondary Purpuras

The conditions with which purpura may be associated have been enumerated on p. 517. It is probable that there is some toxin present which damages the capillaries.

### Hæmophilia

**Definition** A familial disease characterised by a tendency to bleed severely from trivial injuries.

**Etiology** Hæmophilia is usually considered to be due to a deficiency of thrombokinase. It occurs in males but is transmitted by females (Nasse's law). Very rarely it may occur in the female if the mother is a transmitter of the disease and the father is a hæmophilic. These cases may, however, be examples of the hæmorrhagic diathesis or of pseudo hæmophilia. In pseudo hæmophilia an affected father or mother can transmit the disease to children of either sex.

**Clinical Findings** The history frequently suggests the presence of "bleeders" in other male members of the family, and they can often be traced back for more than one generation. The peculiarity is often noted during the second year of life, a trivial injury causing excessive bruising or bleeding if the skin is abraded or cut. Extraction of a tooth may give rise to serious bleeding.

**On Examination** Ecchymoses may be seen under the skin or a hæmatoma felt. A joint may be distended with blood, especially the knee or elbow. The affected joint is painful and the skin over it red.

The temperature then is usually raised to about 100° F. Muscular hæmorrhages may cause pain by nerve pressure. The blood: There may be a hæmorrhagic anæmia. The platelets are not deficient. The bleeding time is normal or slightly prolonged, a prick not causing prolonged bleeding as does a scratch, as the minute wound of the prick is sealed by a mass of platelets. It is therefore quite safe to perform a vein puncture. The coagulation time is prolonged to 5 to 8 times the normal, but it may revert to nearly normal between the attacks.

**Differential Diagnosis.** Hæmophilia must be distinguished from simple obstinate bleeding, such as may occur after a dental extraction. This may affect either sex and there is no history of hæmophilia. In purpura hæmorrhagica the prolonged bleeding time and thrombopenia are characteristic. *Fibropenia* is a rare disease, in which the blood contains no fibrinogen and so will not clot. In the hæmorrhagic diseases of the newborn the bleeding occurs during the first few days of life, usually with melæna.

**Course and Complications.** There is a tendency for the condition to improve with the passage of years. Joints which have been affected with a hæmorrhage may become ankylosed by fibrous tissue.

**Prognosis.** This is always very grave, most hæmophiliacs failing to survive to adult age.

**Treatment.** The patient must be protected from all varieties of external trauma. A small local hæmorrhage may be sometimes arrested by the application of liq. adrenalin. hydrochlor. on cotton wool, or by normal human blood applied on wool after removal of the clots from the wound. If this fails a sterile 1 in 10,000 solution of Russell's viper venom (*Stypven* or *Rusven*) should be applied on gauze plugs. A blood transfusion, using citrated blood obtained from a suitable donor, will restore the coagulation rate of the patient's blood to normal for a period of about 5 days, and must be given before any surgical operation is performed. Hæmarthrosis is treated by elevating the joint and applying bandages wrung out in iced water. In some cases it is necessary to aspirate the joint. Between the attacks all foci of sepsis in the mouth should be removed.

### **Hæmorrhagic Disease of the Newborn** (*Melæna Neonatorum*)

**Definition.** Spontaneous hæmorrhages in newborn infants.

**Etiology.** The cause is hypoprothrombinæmia due to vitamin K deficiency developing a few days after birth and ceasing usually after the eighth day.

**Clinical Findings.** During the first 4 to 10 days of life hæmorrhages occur from the umbilicus, stomach or the intestines. There may also be ecchymoses or bleeding from the urinary tract or vagina. The infant appears fretful, does not take its feeds well, and may be jaundiced. The blood: The platelets are not diminished. The coagulation time and bleeding time are prolonged. There is hypoprothrombinæmia.

**Differential Diagnosis.** Hæmophilia does not often manifest itself at such an early age. Localised hæmorrhages may be due to trauma,

such as a cephalhæmatoma. In Winckel's disease (see p. 86) and Buhl's disease (see p. 86) the condition is one of infective jaundice. Syphilis and umbilical sepsis may give rise to similar symptoms.

**Prognosis** The outlook is grave if the patient fails to respond to adequate treatment.

**Treatment.** *Prophylactic* The mother is given a vitamin K preparation such as Kapon 5 tablets or m. 90 liquid by mouth 12 to 4 hours before the baby is born, and the baby is given 36 drops (m. 18) by mouth on the first day of its life.

*Curative* The baby is given an intramuscular injection of 1 ml Kapon which is repeated after 2 days. In severe cases a blood transfusion of 60 mls of Group O blood should be injected intravenously, and repeated the next day. The injection may be made into the superior sagittal sinus. Intramuscular injections of blood do not appear to raise the prothrombin content of the infant's blood.

#### Hæmorrhagic Thrombocythæmia

This is a rare condition, characterised by a great increase of megakaryocytes and platelets in the bone marrow, and by a blood platelet count of 3 millions or more. Thrombosis of the larger veins of the legs and of the mesenteric vessels is liable to occur, also subcutaneous hæmatomata.

## CHAPTER VII

### THE INFECTIOUS FEVERS

**Introductory.** The diseases included in this section are those which are admitted to isolation hospitals for treatment. Cerebro-spinal fever, acute poliomyelitis and encephalitis lethargica are described in the chapter on nervous diseases.

In practice it is not necessary to send away all such cases to these hospitals, and in view of the risk of "cross infection" (the child being admitted with one disease and contracting another while in hospital), it is better to nurse them at home, provided they can be adequately looked after. Owing to the highly-specialised medical and nursing attention which may be required in cerebro-spinal fever, scarlet fever, enteric fever and diphtheria, it is advisable for patients suffering from these diseases to be admitted to a special hospital.

Certain terms used in describing these diseases will now be defined:—  
*Incubation period*: The time between infection and the first development of symptoms; it is very variable, and the average figures adopted by the Ministry of Health and the Board of Education are given.  
*Prodromal period*: The time between the end of the incubation period and the appearance of the specific rash.  
*Exanthem*: The cutaneous eruption.  
*Enanthem*: Eruptions on mucous membranes, such as Koplik's spots in measles.  
*Quarantine period*: The maximum time during which a person who has been in contact with the infection may develop the disease. It is usually 1 to 2 days longer than the maximum incubation period.  
*Isolation period*: The time the patient must be isolated.  
*Fomites* (fomes = tinder): Articles with which a patient, suffering from an infectious fever, has been in contact, and which convey the infection to another person.

*Notifiable Diseases.* The following diseases are compulsorily notifiable; it will be seen that other diseases than the infectious fevers are included: Small-pox, cholera, diphtheria, scarlet fever, erysipelas, typhus, the enteric group of fevers, relapsing fever, puerperal pyrexia and puerperal fever, plague, cerebro-spinal fever, acute poliomyelitis, acute encephalitis lethargica, acute polioencephalitis, tuberculosis, ophthalmia neonatorum, dysentery, malaria, acute primary pneumonia and acute influenzal pneumonia.

The following can be made locally notifiable in certain areas during an epidemic and for certain periods: Measles, rubella, whooping-cough, chicken-pox, diarrhoea, zymotic enteritis, scabies, pemphigus neonatorum, anthrax, glanders and hydrophobia.

#### Measles

(*Morbilli. Rubcola. (On the Continent Rubcola is synonymous with Rubella)*)

**Definition.** An acute infectious disease, characterised by early catarrh and fever and followed by a typical rash.

**Etiology** The cause is not definitely known but it is probably an ultra microscopic virus. It is suggested that measles encephalo myelitis is due to an independent virus, activated by the measles virus. The disease is spread by direct contact, probably through droplet infection on sneezing or coughing. It is very infectious during the early catarrhal stage before the appearance of the rash.

**Predisposing causes** 1 Age The maximum incidence is between 8 months and 5 years. 2 Absence of a previous attack. Second attacks of genuine measles are very rare. 3 Season Epidemics occur in the winter and spring and are more virulent and extensive in alternate years. 4 Sex climate and race play no part.

**Pathology** There are no special post mortem changes. Death is usually due to complications especially bronchopneumonia.

**Incubation Period** 7 to 14 days usually 10 to 11 days.

**Clinical Findings** The patient is usually a child. A few hours after exposure to infection he may develop symptoms of measles and even a transient morbilliform rash. This is called the "illness of infection". It disappears in a day or so and the measles may then develop at the usual date. The onset is with catarrh of the eyes, photophobia, sneezing, nasal discharge and malaise. A cough, laryngitis or diarrhoea may also be noted. A prodromal rash sometimes occurs which may be scarlatiniform or morbilliform in character.

**On Examination** On the first day the patient appears flushed, the conjunctivæ are injected, the temperature is raised to 99° or 100° F or over. A characteristic enanthem is found, Koplik's spots being seen in over 90% of cases. They are small, bluish white slightly raised spots (due to necrosed epithelium) on a bright red base, about the size of a pin's head, seen inside the cheeks opposite the premolar teeth. The areolæ are often confluent and the spots may spread over the mucous membrane of the cheeks and lips. The temperature often falls to, or below normal on the second or third day (remission of measles) and rises again on the evening of the day before which the rash appears (third day) to 102° F or over and continues to rise until the rash is well out. In an uncomplicated case the temperature falls gradually to normal during the next 48 hours as the rash fades (see Fig 49) but the rash and fever may last for 4 or 5 days. The Koplik's spots usually fade with the appearance of the exanthem.

The rash is first seen on the forehead near the hair and behind the ears. It spreads usually within 24 hours to the face, neck and trunk, and to the limbs, hands and feet including the palms and soles (see Fig 50). The rash at first macular, rapidly forms papules of a dull red colour varying in size from about 2 to 6 mm with an irregular, blotchy appearance. The papules tend to coalesce and form crescentic areas whose colour fades on pressure, the intervening skin being white. Confluence may lead to areas of diffuse erythema especially on the back, and very rarely small vesicles may form on the spots. The rash begins to disappear in 2 to 5 days in the order of its appearance becoming brownish, and leaving some staining which may persist for 1 to 2 weeks. This is followed by a branny desquamation in many cases.

may be slight and abortive, and the skin is often moist and itching. The tongue is usually covered with a whitish-yellow fur. The blood: There is often leucocytosis during the invasion period, followed by leucopenia with a relative increase in the large lymphocytes. The

urine: Febrile albuminuria is frequently found and Ehrlich's diazo-reaction is usually given during the febrile period. There is frequently some bronchitis.

**Varieties:** Variations from the typical case may occur. These include: 1. *Morbilli sine morbillis*: No rash follows the typical early symptoms. This occurs particularly after the administration of serum, which has been given with a view to allowing an attenuated attack to occur. 2. *Hæmorrhagic or black measles*: This is very rare and fatal. Hæmorrhages occur from the mucous membranes and into the skin. The cases so diagnosed were probably small-pox. Petechial hæmorrhages into the spots are not of grave significance. 3. *Toxic measles*: The patient is overwhelmed with toxæmia, characterised by delirium, tremors, hyperpyrexia and death. 4. *Suffocative measles*: There is marked dyspnoea from

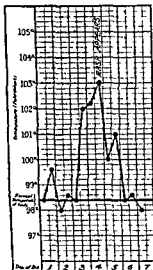


FIG. 40. TEMPERATURE CHART IN MEASLES.

the onset, with râles heard all over the lungs.

**Differential Diagnosis.** During the invasion period the disease may be mistaken for a cold, influenza, laryngeal diphtheria, bronchitis or enteritis.

The age of the patient, history of exposure to infection and, above all, the presence of Koplik's spots are of value in making a diagnosis. The rash may be mistaken for that of rubella, the prodromal rash of small-pox or scarlet fever, syphilitic roseola, a serum rash, septic rash, intestinal rash, post-vaccinal eruption, drug rashes such as those due to copaiba, chloral, turpentine, antipyrin, etc., or for that of typhus fever.

**Course and Complications.** The typical course has been described above, but this may be seriously modified by complications. The most important of these are: Bronchitis, bronchopneumonia, laryngitis, laryngeal ulceration, pleurisy, empyema, diphtheria, cervical adenitis, blepharitis, corneal ulceration, panophthalmitis, herpes facialis, otitis media, cancrum oris and noma pudendi. Convulsions are not uncommon; encephalitis, myelitis, cerebellar syndromes, toxic psychoses, osteomyelitis and nephritis are rare. Nervous complications are most likely to occur between the third and fifth days of the exanthem, or the eighteenth and twentieth days of convalescence. Enteritis ranks next to bronchopneumonia as the most serious complication. It may occur during the prodromal stage, in the eruptive period, or after the rash has faded. Bronchial gland or pulmonary tuberculosis or general adenitis may develop as sequelæ. Relapses are extremely rare.

**Prognosis.** This is very good apart from complications, but owing to the frequency of bronchopneumonia, measles causes a high mortality rate in infants, especially amongst the poor.

**Treatment Prophylactic** Pooled convalescent serum, previously tested for sterility and obtained from donors free from infection with tuberculosis and syphilis, can be administered to contacts. It is injected intramuscularly into the vastus externus. It is put up in ampoules of 5 mls. The dose is 5 mls for a child up to the age of 5 years, and over this age the dose is doubled. Complete protection is afforded if the injection is given during the first 5 days of the incubation period, such protection lasting for 3 weeks. If given later than 5 days after exposure to infection, an attenuated attack may occur, with subsequent life long protection. Adult measles serum can also be given. This is obtained from people who have had measles not more than 10 to 20 years previously. A dose of 10 mls is given intramuscularly up to the age of 5 years, and over this the dose is doubled. The serum should be given within 5 days of exposure, when the immunity produced lasts about 3 weeks. An attenuated attack may result if half the dose is given within the first 5 days of the incubation period, or if the full dose is given between the sixth and ninth days. Adult serum is not nearly so certain in its action as is the convalescent serum. A placental extract, obtained from the placenta of women who have had measles not more than 10 to 20 years previously, has also been tried, but the results are not very satisfactory. Further, no lasting immunity is conferred. The dose is 2 mls for an infant and 6 mls for a child over 5 years, injected intramuscularly into the vastus externus. A second dose is repeated on the next day but one. The immunity produced lasts about 3 weeks. It will prevent an attack if given during the first 5 days of the incubation period, and attenuate it if given later in the incubation period.

**Quarantine period** This is 3 weeks for contacts. They need not be kept from school for the first week, but should be isolated from all children under the age of 5 years, and subsequently should be examined daily for early signs such as catarrh, fever, Koplik's spots and a prodromal rash.

**Isolation period** This is 2 weeks from the appearance of the rash.

**Curative** The patient must be put to bed immediately, and kept there for 2 to 3 days after the temperature is normal. The temperature

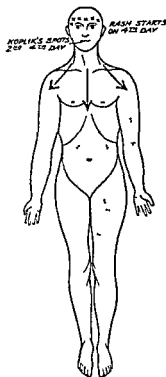


FIG. 50. DIAGRAM OF RASH IN MEASLES

Rash spreads downwards back and front, arms, legs, palms and soles for 24 to 36 hours and fades about 2 days later, leaving some staining and branny desquamation. The crosses indicate the usual site of the first appearance of the rash.

of the room should be maintained day and night at 60° to 65° F.; adequate ventilation must be ensured, and if there is any bronchitis the atmosphere should be moistened with a steam kettle. The bed should not face the light owing to the photophobia. Milk diet and dextrose orangeade only must be given while the temperature is raised. The bowels should be kept open daily with liq. magnes. bicarb. m. 120 for a child of 5 years, as necessary.

If there is high fever or much irritation, the skin should be tepid sponged once or twice a day. If the rash does not come out well, a hot sponge and hot bottles should be applied. The eyes should be bathed with warm boracic lotion 2 or 3 times daily and vaseline put on the lids. Argyrol (argent. proteinas mite B.P.C.), 25%, may also be dropped into the eyes twice daily. The inside of the mouth should be cleansed before and after meals with cotton wool dipped in  $H_2O_2$  (10 vols) diluted 8 times with water. The patient should be encouraged to blow his nose from time to time. Excessive bedclothes should be avoided. Convalescent serum treatment is probably of no value after the onset of the disease. Encephalomyelitis is treated by lumbar puncture to relieve pressure, and by the intravenous injection of 20 mls of serum from a patient convalescent from measles encephalitis. In order to prevent or lower the incidence of bronchopneumonia and otitis media, sulphanilamide may be given for 5 days in doses varying with the age of the patient, e.g., 1 G. t.i.d. for a child of 5 years. This may result in fever and the appearance of irritating rashes, urticarial, scarlatiniform or morbilliform.

No rigorous disinfection of the room is necessary after measles. Sheets, etc., should be boiled and the mattress aired for a couple of days. The patient should have a bath and the hair washed before mixing with others. The most important complication is bronchopneumonia, for the treatment of which see p. 140.

### German Measles

(*Rubella. Röteln*)

**Definition.** An acute infectious disease characterised by fever, enlargement of glands and a typical rash.

**Etiology.** The cause is not known. Rubella is spread by direct contact, probably by droplet infection from the naso-pharynx.

**Predisposing causes:** 1. Age: Children and young adults. 2. Season: The greatest number of cases develop in the first half of the year. 3. Absence of a previous attack: Second attacks are very rare.

**Pathology.** There is no morbid anatomy, for death practically never occurs.

**Incubation Period.** 5 to 21 days, usually 17 to 18 days.

**Clinical Findings.** The patient is usually a child or young adult. Often the first symptom noticed is the rash, although there may be headache, malaise, sore throat and nasal catarrh for a day or so earlier, or stiffness in the back of the neck, due to the enlarged glands. An acute onset with a rigor, stiff neck and occipital headache may occur.



**On Examination** During the invasion stage the temperature is usually raised to  $99^{\circ}$  or  $100^{\circ}$  F., the eyes are somewhat pink and enlarged glands are felt at the back of the neck. There are no prodromal rashes. The rash appears usually on the second or third day, first on the forehead and behind the ears, then the face, trunk and limbs are involved (see Fig 51). The rash consists of small macules and papules nearly circular and about 1 to 4 mm in diameter. The colour is pink or light red. The rash disappears from the face

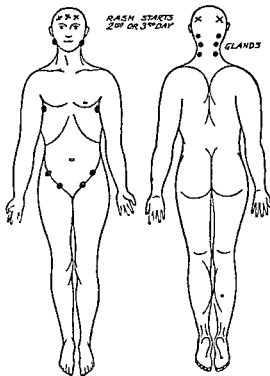


FIG 51 DIAGRAM OF RASH IN GERMAN MEASLES

Rash spreads rapidly to face neck trunk and limbs. Legs are less affected than arms. The rash usually fades in 36 hours and leaves no staining. The crosses indicate the usual site of the first appearance of the rash. The black circles indicate glands.

in 12 to 24 hours and there is no staining but slight desquamation may occur. The rash is more confluent on the trunk and may be almost scarlatiniform, on the extremities it is more morbilliform, irregularly blotchy in character and frequently involves the palms and soles. It usually fades completely in 36 hours. The temperature falls by remissions and reaches normal on the second or third day after the appearance of the rash. There may be very slight or no pyrexia (see Fig 52). The glands. The following groups especially are enlarged. Suboccipital posterior cervical, those at the angle of the jaw, the mastoid axillary, and inguinal glands. Suppuration does not occur. The conjunctivae may be slightly inflamed and the spleen just

palpable. The blood: Lymphocytosis is usually present before the rash comes out, later there is leucopenia. The urine is usually normal and no diazo-reaction is obtained.

**Differential Diagnosis.** On the second day of the rash there may be difficulty in distinguishing the disease from scarlet fever owing to the rash having faded from the face, leaving a circum-oral pallor, and being confluent on the trunk. The chief points indicative of rubella are: The enlarged glands, especially behind the neck and in the axillæ, the absence of throat signs, the slight degree of pyrexia, the slight constitutional disturbance, and later the absence of pin-hole peeling. Diagnosis from measles may also be difficult, but the absence of severe coryza and Koplik's spots during the invasion period of rubella, and its characteristic rash and enlarged glands serve to differentiate it. Drug, enema and toxic rashes may also simulate rubella, but are usually differentiated by the history and absence of typical clinical findings.

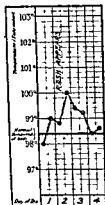


FIG. 52. TEMPERATURE CHART IN GERMAN MEASLES.

**Course and Complications.** The course is as described above. The patient is generally quite well in a week and there are usually no complications, although bronchitis, stomatitis and severe muscular pains sometimes occur.

**Prognosis.** This is invariably good.

**Treatment.** *Prophylactic.* There is no special preventive treatment. *Quarantine period* for susceptible contacts is 21 days. *Isolation period.* The patient should be isolated for 7 days from the appearance of the rash.

*Curative.* The patient should be put to bed and kept there for 2 days after the rash has disappeared. No special treatment is required beyond that applicable to a mild degree of pyrexia. Disinfection of the patient and the room is as for measles (see p. 528).

### Scarlet Fever (*Scarlatina*)

**Definition.** An acute infective disease characterised by fever, sore throat, a typical rash and subsequent desquamation.

**Etiology.** The disease is generally considered to be caused by the streptococcus scarlatinæ, a hæmolytic organism of which there are twenty-eight Griffith types pathogenic to man. Whether scarlet fever or simple streptococcal infection occurs as the result of invasion with a hæmolytic streptococcus, depends upon the amount of erythrogenic toxin produced by the streptococcus, and on the specific resistance of the host to that toxin. The erythrogenic toxin is responsible for the rash, congested fauces and red tongue. Infection is spread either by direct contact, probably by droplets, or indirectly by fomites such as clothes, toys and books, by milk, feeding utensils, by attendants on patients and by carriers. The desquamated papules are

not infective. **Predisposing causes** 1 Age The majority of cases occur between the ages of 5 and 10, young adults also are often affected 70% of infants, according to the Dick test, are susceptible between the ages of 1 and 2, but only 18% of adults after the age of 20 2 Season Epidemics tend to occur in recent years in January, few cases are seen in the summer. 3 Climate The disease occurs chiefly in the temperate zones 4 Absence of a previous attack Second attacks may, however, occur 5 Convalescence from diphtheria

**Pathology** There is engorgement of the fauces, dilatation of the cutaneous capillaries, and cervical adenitis Inflammation may be

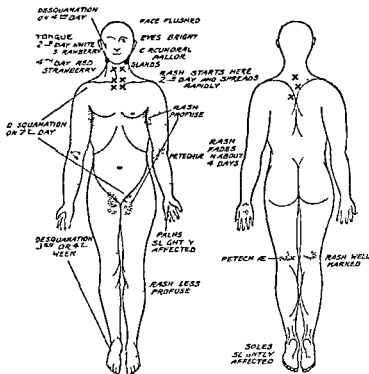


FIG. 53. DIAGRAM OF RASH IN SCARLET FEVER

The crosses indicate the usual site of the first appearance of the rash. The black circles indicate glands

found in the pericardium, endocardium and joints of the fingers and wrists In rapidly fatal cases the patient dies from toxæmia In other cases death may be due to complications

**Incubation Period** 1 to 8 days usually 2 to 3 days

**Clinical Findings** The patient is usually a young child who is suddenly taken ill with headache, sore throat and vomiting

**On Examination** The child has a brightly flushed face and a pale zone around the mouth (circum oral pallor) The eyes are bright but not injected, the tongue has a thick yellowish fur, and the tonsils are red, and may show some exudate A few red spots may be seen on the soft palate The temperature is raised to about 102° F, and the pulse

is frequent, about 180. The rash appears on the second day, first on the neck and upper part of the front of the chest, spreading in a few hours to the trunk and limbs. It is especially well marked in the axillæ, groins, flexures of the elbows and knees and inner side of the thighs. The backs of the hands and feet and to a lesser degree the palms and soles are involved, but except for the flush, the face is not usually affected (see Fig. 53). The rash consists of small, bright red points on a red base (punctate erythema) the whole fading on pressure. The skin feels dry and hot. Definite staining may be left after the rash has faded, which usually takes 4 days. Small petechiæ may then be seen on the flexor aspects of the elbows and knees.

Desquamation begins about the fourth day, first on the face, where it is very fine and causes the "powder and rouge" appearance. About the seventh day it is seen on the side of the neck, below the clavicles and above the pubes. It is called "pin-hole" peeling as small holes form over the sites of the red points of the rash. As these enlarge and coalesce flakes are detached. In the third and fourth weeks the hands and palms peel, the heels being affected last of all. The tongue presents characteristic changes, on the second day red papillæ show through the creamy fur ("white strawberry" tongue). The tip and edges then begin to peel and on the fourth day the "red strawberry" or "ripe raspberry" tongue is seen, the whole tongue being bright red with prominent papillæ. In 80% of cases a throat swab shows hæmolytic streptococci. Enlarged glands are present below the angles of the jaw, and these may suppurate.

**The temperature:** This rises rapidly at the onset, falling by lysis in a mild case on the fourth or fifth day, to reach normal about the eighth day or earlier (see Fig. 54). **The blood:** Leucocytosis and eosinophilia are usually present during the pyrexial stage. **The urine:** Febrile albuminuria is often noted, and the diazo-reaction may be given.

**Varieties.** 1. *Abortive.* The rash may be very slight or only seen on certain parts of the body, but it is followed by desquamation, and some complication may ensue.

2. *Malignant or Toxic.* Constitutional disturbances include prostration, hyperpyrexia, vomiting, diarrhœa, delirium, insomnia, and exhaustion; death results in 1 or 2 days. The rash is not usually well marked.

3. *Septic.* Severe tonsillar or palatal lesions with ulceration and necrosis are seen. Otorrhœa may ensue, cervical adenitis and cellulitis are present, and there may be hæmorrhage from erosion of an artery.

4. *Hæmorrhagic.* This rarely occurs, and is very fatal. Hæmorrhages are seen in the skin and from the mucous membranes.

5. *Surgical.* This follows injuries, especially burns and operations, particularly on the naso-pharynx. The infection may enter through the throat or through the skin lesion.

6. *Puerperal.* This is not a special variety of scarlet fever. A woman during the puerperium may contract scarlet fever, but is probably not especially susceptible.

**Differential Diagnosis.** During the invasion period scarlet fever

must be diagnosed from tonsillitis and diphtheria. A swab should be taken from the throat if exudation is present. The rapid onset of the rash usually serves to establish the diagnosis. The rash must be diagnosed from the following: Measles rash, rubella rash, septic rashes, eczema rash, flannel rash, prodromal rash of small pox, erythema due to influenza, drug rashes due to belladonna, salicylates, copaiba, and quinine. The distinguishing characters of the rashes due to the infectious fevers are considered under their separate headings. The following additional tests are of value.

**The Schultz Charlton Test** or extinction sign. One fifth of a mil of a 1 in 10 dilution of scarlatinal antistreptococcus serum is injected intradermally into the abdomen of the patient where the rash is well developed. If the disease is scarlet fever, a circular area of blanching occurs in about 6 to 8 hours lasting for about 3 days. No blanching results if the rash is due to other conditions. A positive result is obtained in about 90% of scarlet fever cases during the first 3 days of the rash.

**The Dick Test.** Two tenths of a mil of the appropriate dilution of the scarlet fever streptococcal toxin are injected intradermally, and the cutaneous reaction compared with that resulting from a similar injection of the control heated toxin. The test is positive during the first 3 days of scarlet fever in about 90% of cases and becomes negative in about 10 days' time or later. If the test is negative at the early stage, the disease is probably not scarlet fever.

**Course and Complications.** The course depends on the severity of the disease and the presence of complications. It is also modified by treatment with anti scarlatinal serum. During the fourth or fifth week of the disease a relapse may occur, which is a repetition of the original illness. The most important complications are Nephritis. This is liable to occur about the twenty first day of the disease (see also pp 447, 450) focal nephritis may occur in the first week. Rheumatism. Arthritis may appear at the end of the first week during the second week, or later. The fingers, wrists, shoulders, ankles and knees are most often affected. Suppuration is very rare. Muscular rheumatism is not so common. Other complications include otitis media, meningitis, cerebral abscess, endocarditis, pericarditis, cervical adenitis, persistent rhinitis, bronchitis, ulcerative stomatitis, vaginitis and diphtheria. Perforation of the pharyngeal wall may lead to surgical emphysema, perforation of the palate may also occur. Embolic or thrombotic gangrene of the extremities has also been recorded.

**Prognosis.** This varies with the type of disease. Toxic and hæmorrhagic cases are usually rapidly fatal and septic cases frequently

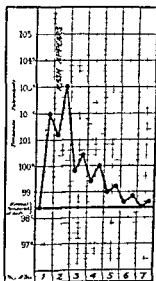


FIG 54. TEMPERATURE CHART IN SCARLET FEVER

die. The prognosis is good in simple scarlet fever, although sequelæ may result from complications such as nephritis, endocarditis, or otitis media, which may prove fatal several years later.

**Treatment.** *Prophylactic.* Contacts may be tested by the Dick reaction to see if they are susceptible to scarlet fever. The negative reactors need not be isolated. It is also advisable to test patients before operations on the naso-pharynx. *Quarantine period.* Positive reactors should be placed in quarantine for 7 days. They may be immunised passively by the intramuscular injection of 10 mils of concentrated scarlet fever streptococcus antitoxin, but this of course renders them sensitive to horse serum, and so liable to subsequent anaphylaxis. Workers in infectious fever hospitals should be immunised actively by means of injection of scarlet fever "prophylactic" (toxin). Immunisation by this method takes 3 to 4 weeks, and hence will not afford immediate protection, but it will often check recurrent attacks of scarlet fever in a school. As scarlet fever is so benign at the present time, mass immunisation of children does not appear necessary. *Isolation period.* The minimum isolation period for a simple case is 4 weeks, but the patient must be isolated while there is any discharge from the nose, ears, vagina or a wound, and until the nose and throat are apparently healthy. Children must not return to school for 2 weeks after release from isolation. A hæmolytic streptococcus is present in the throat in many cases on discharge from hospital and peeling is often not completed.

*Return cases* are fresh cases of scarlet fever resulting from infection by the convalescent patient after he has been freed from isolation. In London the return case rate is about 3%. The infective agent in such cases is probably still present in the nose or throat. A throat swab, however, which is negative as regards the hæmolytic streptococcus does not mean that the patient is free from infection.

*Curative.* The patient must be kept in bed for at least 2 weeks. The temperature of the room should be maintained at about 62° F., and the room should be well ventilated; if possible a coal fire should be burning, so that swabs contaminated by discharges can be destroyed at once. A sheet hung over the door, moistened with carbolic lotion, serves to keep off intruders, but does not prevent spread of infection. A flannel night-gown should be worn. Diet: During the febrile stage. Milk, dextrose-orangeade, barley water and plenty of water should be given, with subsequent additions of bread and butter, honey, cream, potatoes, porridge, bananas, green vegetables, milk soups, eggs, etc. A fish diet can usually be taken at the end of the first week if the patient has had serum treatment, and an ordinary diet during the second week.

The patient should be washed or bathed daily with soap and water, and may be sponged in addition during the febrile period. Hot water bottles help to bring out a poorly marked rash. Forceful spraying of the throat is deprecated, as it tends to spread infection to the middle ear. A gargle of glycerin, thymol. co. (B.P.C.) m. 60 to a glass of warm water may be used 2 or 3 times a day, and the mouth should be cleansed after each feed with some antiseptic, such as glycerinum boracis on a

**swab** The nose should be swabbed out as required. The bowels should be opened daily with salines, or a laxative, such as syrup *ficorum* m 60 to 120 or *Cascara Lvacuant* m 30 to 60. In order to try to prevent nephritis, alkalis may be given daily to keep the morning urine alkaline. Equal parts of *sod bicarb* and *pot cit* are used, in doses up to gr 30 of each t d s for a child under 7 years, and gr 60 of each t d s over the age of 7 years. Many observers have found that the incidence of nephritis is unaltered by the alkali treatment. Specific treatment in the form of the injection of concentrated scarlet fever streptococcus antitoxin is now largely used, for simple as well as severe cases of scarlet fever. In mild cases the serum usually causes the temperature to fall rapidly by crisis, but it is doubtful whether the incidence of septic complications is lowered by its use, although toxic complications especially rheumatism, appear less frequent. The disadvantage is that the patient becomes sensitised to horse serum and runs the risk of subsequent anaphylaxis if serum is given later for another disease. For a moderately severe case 30 to 40 mls (6,000 to 8,000 U S A units), and half this dose for a child are injected intramuscularly. The "globulin modified" antitoxin containing 9,000 units in 8 mls can also be used. A single injection is usually sufficient. If the injection is given intravenously or intraperitoneally half the dose is required. Alternatively, 30 mls of convalescent serum can be injected intramuscularly, and this avoids sensitising the patient to horse serum. Sulphapyridine, 1 G every 4 hours, should also be given for 3 to 4 days to adults, and half the dose to children. Before giving antitoxin intravenously, intramuscularly or intraperitoneally, conjunctival and intradermal tests for horse serum sensitivity should be applied. One drop of horse serum, diluted 1 in 10 with normal saline, is instilled into the lower conjunctival sac. A positive reaction is shown by lachrymation, irritation, and redness within 20 minutes, and this constitutes an absolute contra indication to the intravenous route. If this is negative the intradermal test is performed by injecting intradermally 0.2 ml of antitoxin, diluted 1 in 10 with saline. If an urticarial wheal develops, the patient is sensitive to serum, and must be desensitised by injecting small doses subcutaneously. First  $\frac{1}{2}$  of a minim, diluted with saline, is injected, and the dose is doubled every half hour until m 15 has been given. A solution of liq adrenalin hydrochlor should be at hand, and 0.5 ml injected intramuscularly if there is a reaction.

**Septic Cases** Fomentations should be applied to the neck, and the concentrated antitoxin (scarlet fever) is injected as detailed above. A further course of Sulphapyridine should be given at the onset of septic complications. Stimulants may be necessary, such as brandy m 60 t d s, or Coramine (nikethamidum B P Add) 1.5 ml subcutaneously t d s for a child of about 5 years.

**Toxic Cases** Twenty mls (4,000 U S A units) of serum should be given intravenously, warmed to body temperature, but not diluted. At the same time 40 mls of serum should be injected intramuscularly. It may be necessary to give another 40 mls of serum intramuscularly.

12 to 24 hours later. An alternative method consists in the direct intramuscular injection of 100 mils of whole blood, obtained from a convalescent case of scarlet fever. Stimulation by the administration of brandy should be given, and the patient kept warm with hot bottles. Complications are treated as follows: Nephritis: This is treated as described on p. 449 for acute diffuse glomerulo-nephritis. Arthritis: The joints should be wrapped in wool, and a mixture given containing Sod. salicyl. gr. 10, sod. bicarb. gr. 20, syr. aurant. m. 20, aq. ad fl. oz. 1. Fl. oz. 1 t.d.s. Wool sheets or blankets should be used. Cervical adenitis: Antiphlogistine (kataplasma kaolini B.P.) is applied to the neck, and the glands incised when suppurating. Otitis media: Dry heat is applied to the ear, by covering the ear with wool, the patient lying on a small rubber hot-water bottle. If this does not relieve the pain, fomentations are used. An aural surgeon should always be consulted and the drum incised when necessary; if the drum has perforated a few drops of peroxide of hydrogen should be instilled, and the ear mopped out with cotton wool. Rhinitis: A swab should be taken, and if virulent Klebs-Loeffler bacilli are found, 8,000 units of diphtheria antitoxin are given intramuscularly. Faucial diphtheria requires serum treatment also, but in larger doses (see p. 540), and without awaiting the virulence test.

Discharge from isolation: The patient should have a hot bath, and the hair should be washed the night before he is discharged; he should then, if possible, sleep in a fresh room in a clean bed. The sick room must be thoroughly disinfected.

#### Fourth Disease

##### (*Filatow-Duke's Disease*)

This is described as a disease distinct from scarlet fever, measles and rubella. A previous attack does not protect against either of the latter diseases. The cause is unknown. The incubation period is 9 to 21 days. The rash is scarlatiniform, but there is no circum-oral pallor. Desquamation occurs. The cervical, axillary and inguinal glands are slightly enlarged, and there is mild pyrexia. The patient should be isolated for 8 weeks.

#### Fifth Disease

##### (*Erythema Infectiosum*)

This disease is very rare in England. The incubation period is 5 to 10 days. The rash is polymorphous, and is seen on the cheeks, arms and legs, chiefly on the extensor surfaces, and slightly on the trunk. It may be morbilliform, scarlatiniform or annular, and usually fades in a week. There is slight pyrexia.

#### Diphtheria

##### (*Membranous Croup*)

**Definition.** An acute infective disease characterised by localised membrane formation and toxæmic disturbances.



**Etiology** Diphtheria is caused by the *Coryne bacterium diphtheriæ* (*B. diphtheriæ* or *Klebs Loeffler bacillus*). Three types are described: the *gravis*, which ferments starch, the *intermedius* and the *mitis*, which are non fermenters of starch. The evidence now available does not support the view that the *gravis* type is associated with a highly toxic variety of diphtheria. *Predisposing causes* 1 Age Chiefly in children 1 to 15 years 2 Season Autumn and winter, epidemics occur 3 Other diseases Scarlet fever and measles 4 Sex Both sexes are equally affected in childhood, later, females predominate 5 Climate Chiefly in temperate zones

Diphtheria confers only short lived immunity and second attacks are not infrequent. The disease is spread by direct contact, through droplet infection, as in coughing and kissing by fomites, especially pencils which are sucked, or feeding utensils and handkerchiefs, and by milk and carriers. The cow's udders may be infected.

**Pathology** The membrane is formed of serum, fibrin, necrosed cellular tissue and organisms. It may occur in the pharynx, nose, larynx, trachea and rarely in the œsophagus and stomach or on the skin. Fatty degeneration of the myocardium may rapidly develop. Bronchopneumonia is at times due to inhaled diphtheria bacilli. The kidneys may show acute interstitial or glomerulo tubular nephritis. Degeneration of the medullary sheath of peripheral nerves may be found.

**Incubation Period** 2 to 10 days, usually 3 to 4 days.

**Clinical Findings** There are three main types of the disease: faucial, laryngeal and nasal, these will be separately considered.

**Faucial Diphtheria** The patient is usually a child who feels ill and may have headache or limb pains. Frequently there is no complaint of the throat. Vomiting or diarrhoea may occur early.

**On Examination** The child looks ill and pale in severe cases. The exudate in the early stages is seen as isolated greyish yellow patches on the tonsils or fauces. These spread and coalesce to form a membrane which may extend to the soft palate, pillars of the fauces, uvula and posterior wall of the pharynx. Swelling of the surrounding tissues in the throat and neck is marked in severe cases, and the membrane may become greenish black and emit an offensive odour. It can be stripped with some difficulty, leaving a bleeding surface. The submaxillary and cervical glands enlarge to a variable degree, and may form a collar ("bull neck"). The tongue is usually furred. The temperature typically is only moderately raised, 99° to 101° F, and returns to normal in 3 or 4 days. The pulse, however, is unduly rapid, often 110 to 120. The blood. There is usually a leucocytosis. The blood sugar curve is of the diabetic type in toxic cases. The urine. Temporary albuminuria is frequent.

**Laryngeal Diphtheria** This may occur independently or complicate faucial diphtheria. It is rare in adults. In children it is a cause of "croup." The child becomes hoarse, and has paroxysmal attacks of dyspnoea and "croupy" cough.

**On Examination** If the laryngeal obstruction is marked signs of

deficient pulmonary ventilation are evident, such as cyanosis, laboured breathing with the accessory respiratory muscles in action, intercostal recession, laryngeal excursion and weak air entry into the lungs. The membrane may extend to the trachea or main bronchi.

*Nasal Diphtheria.* This is often associated with faucial diphtheria, or may occur independently. It is characterised by a watery or mucous nasal discharge which is usually blood-stained and often unilateral. The membrane is generally in the posterior part of the nose, but may be visible in the nostrils.

*Other varieties of diphtheria include:* Aural, usually of the external ear. Conjunctival. Vulval or anal. Cutaneous. A membrane may form over wounds or the lesion may resemble eczema or impetigo; a diphtheritic whitlow may occur. Hæmorrhagic. Severe cases of diphtheria with hæmorrhages into the skin and bleeding from mucous membranes. Bacteriological. Klebs-Loeffler bacilli are found in the throat, but no membrane forms.

*Differential Diagnosis.* *Faucial Diphtheria.* This must be differentiated from follicular tonsillitis, quinsy, the appearance of the throat after tonsillectomy, thrush, Vincent's angina, agranulocytic angina, syphilis of the tonsil, scarlet fever, mumps and bronchopneumonia.

The throat must be examined as a routine in every patient. The clinical diagnosis of diphtheria is suggested by the membrane, the cervical glandular enlargement, the slight pyrexia and the presence at times of albuminuria. The bacteriological report does not exclude the disease if no diphtheria bacilli are found, and clinical cases should be treated as such without awaiting bacteriological confirmation.

*Laryngeal Diphtheria.* This must be diagnosed from other causes of "croup" (see p. 119), from the onset of measles or from bronchopneumonia. Search should be made for Koplik's spots, and a swab should be taken from the throat.

*Nasal Diphtheria.* This is most likely to be mistaken for nasal discharge from some other cause, such as a foreign body impacted in the nose. A swab should be taken both from the nose and fauces.

*Course and Complications.* The course of faucial diphtheria depends very largely on the treatment. Serum administered on the first day almost invariably produces rapid improvement: if given after the fifth day it has little effect in checking the disease, but it should nevertheless be given. Laryngeal diphtheria is very serious unless treated early with serum. Nasal diphtheria is usually mild, but may spread to the fauces and be complicated by paralyses. Relapses in diphtheria are rare, but the membrane may re-form after the third week: the relapse is not usually grave.

The important complications are: *Circulatory Failure.* This may be due to (a) Myocardial degeneration, occurring in the second week or later. Clinically the temperature falls, the patient becomes restless, there may be præcordial pain and vomiting; the pulse rate either quickens or slows. Variations in the heart sounds are due to gallop rhythm, premature systoles, auricular flutter or fibrillation. The liver

may become engorged, and the extremities cold (b) Peripheral vascular failure (direct toxic effect) This occurs early, during the first week of the illness. The patient becomes pale, collapsed, cold, moist and there may be slight general œdema. The condition resembles shock, and the heart may beat forcibly until a short time before death. (c) Vagal neuritis may be responsible for cardiac failure in some cases.

*Paralyses* (a) Palatal, in the second week. There is a nasal voice and nasal regurgitation. (b) Ocular, in the third and fourth weeks. Extrinsic usually of the VI nerve, with squint and diplopia. Intrinsic, the pupils usually react to light and accommodation, but there is difficulty in reading small print unless the patient is myopic. (c) Pharyngeal, in the sixth week. There is dysphagia, and coughing attacks may occur on swallowing. (d) Laryngeal, in the sixth week. There is usually adductor paralysis, with hoarseness. Less frequently the abductors are affected, with stridor. (e) Diaphragmatic and Inter costal, in the sixth week. There is dyspnoea, reversal of abdominal movements with respiration, intercostal recession and possibly massive collapse of one lung. (f) Limbs, Trunk and Neck, in the sixth to eighth weeks. Weakness of muscles, loss of deep reflexes and blunting of sensation may be found.

The paralysed muscles usually recover in about 4 weeks. Complete recovery is the rule.

Other complications include Bronchopneumonia, otitis media, mastoiditis, nephritis, endocarditis with myocarditis, cerebral embolus derived from a thrombus in the apex of the left ventricle producing hemiplegia or arterial embolus resulting in gangrene of a limb.

*Prognosis* This has been greatly improved by the early use of serum, but 2 to 10% of all cases still prove fatal. The prognosis is more unfavourable if the membrane is extensive, and in very young children. A fall in the diastolic blood pressure is a bad sign. The mortality rate is still high for laryngeal and naso-facial diphtheria. Hemorrhagic cases are usually fatal. Paralyses are rare in laryngeal diphtheria.

*Treatment Prophylactic* Diphtheria is a preventable disease and can be eradicated from a community provided that 80% of children in the age group of 1 to 15 years are efficiently immunised. The susceptibility of individuals can be determined by the Schick test. Two tenths of a mil. of toxin (1/50 M.L.D.) are injected intradermally and the result is compared with that of the control heated toxin, injected into the other arm. Positive reactors can be actively immunised. It is advisable to immunise all children irrespective of their Schick reaction. It is best done at the age of 1 year. Various preparations are available, but those mostly in use are toxoid, antitoxin floccules (T.A.F.) for adolescents and adults and alum toxoid (A.P.T.) for children. Three injections of 1 mil. of T.A.F. are given intramuscularly at intervals of 2 weeks. With A.P.T. a single intramuscular injection of 1 mil. may be given, or preferably 2 doses of 0.5 mil. at intervals of 4 weeks for children under 8 years, and for children over the age of 8, 0.25 mil. followed in 4 weeks by 0.5 mil. if there is no reaction. The injections should be given into opposite arms. A preliminary Moloney

test should be done in a child over the age of 8 years to determine the sensitivity to toxoid before A.P.T. is injected. This consists in the intradermal injection of 0.2 mil. of a 1 in 10 dilution of the toxoid. A positive reaction is shown by the development in 48 hours of an erythematous zone 20 to 30 mm. in diameter. If the Moloney test is positive smaller doses of A.P.T. should be used, 0.1 mil. followed by 0.4 mil. 4 weeks later. The immunity produced by T.A.F. does not develop for about 3 months, but probably lasts over a year. Immunity may be produced by A.P.T. in two weeks, and the child is not sensitised against serum. *Quarantine period.* Contacts of a case of clinical diphtheria with negative throat swabs and a negative Schick reaction and no sign of diphtheria elsewhere need not be isolated. If the Schick reaction is positive the contact must be isolated for 14 days and kept under close observation. Immunisation with A.P.T., as described above, should be begun. If the nose or throat culture is positive, the individual may be a carrier of virulent or avirulent bacilli, or he may be incubating clinical diphtheria; the latter will be determined in a day or so, the former can only be decided by inoculation of the organisms into a guinea-pig. Immunisation with A.P.T. should be carried out at once. Prophylactic inoculation of anti-diphtheritic serum confers passive immunity for about 3 weeks, but it is not justifiable in view of the subsequent danger of anaphylaxis. If the contacts are under supervision, serum can be given on the first day of the disease, should it develop. Chronic carriers of virulent bacilli may be treated with stock or autogenous diphtheria vaccines, in doses of 5 to 10 millions every 5 to 7 days, or have their tonsils or adenoids removed if they are infected. *Isolation period* The patient is isolated for 4 to 6 weeks, until the local lesions have healed and there are no discharges, and 3 consecutive negative swabs of the nose and throat have been taken at intervals of 3 days.

*Curative.* The patient must be carried to bed directly the disease is diagnosed and not allowed to walk, in view of the possibility of myocardial weakness. He must be kept in bed 4 to 8 weeks, for the first 2 weeks only one pillow is allowed, and the patient is put on "absolute rest," owing to the danger of heart failure. A second pillow is then given and gradually he is allowed to sit up. Directly the patient is in bed, and a swab has been taken, anti-diphtheritic serum is injected. The amount depends upon the severity of the case and the duration of the disease, rather than on the age of the patient. A double dose is required if the disease has been present over 48 hours. For a mild case affecting the tonsils and fauces, 8,000 to 16,000 units are given for an adult or child, injected intramuscularly into the middle of the outer side of the thigh. Concentrated serum containing 1,500 units per mil. or highly concentrated serum containing 2,000 to 2,500 units per mil. is obtainable. For a moderate case affecting the tonsils, soft palate and uvula, 24,000 to 40,000 units are given intramuscularly. In nasal cases 6,000 units are required. Good effect is shown by improvement in the general condition of the patient, who becomes brighter, and by cessation of spread of the membrane. A second injection should be given in 12

hours if there is no improvement or if the membrane has spread. In very severe cases combined intravenous and intramuscular injections of massive doses of serum are given such as 70 000 to 200 000 units. The serum is warmed to body temperature but not diluted before intravenous injection and a prophylactic intramuscular injection of *liq adrenalin hydrochlor* 2 mls is given at the same time to prevent anaphylactic shock. After an intravenous injection which must be given very slowly a careful watch should be kept on the patient and if the blood pressure falls Pitressin or Veritol should be injected as described below. A simultaneous intravenous injection of 20 G of dextrose in 40 mls of normal saline with subcutaneous or intravenous injection of 10 units of insulin is of value in very toxic cases and should be given twice a day for the first two days. If intravenous injection is impossible 20 mls of 10% dextrose may be given intramuscularly in the buttock. Serum sickness as shown by oedema rashes fever and joint pains is liable to occur about 5 to 12 days after the injection but is less frequent in children and with the use of concentrated serum. Should it occur an intravenous injection of 5 mls of calcium gluconate (B P Add) should be given. Local treatment to the throat is dangerous and should be avoided. All discharges should be received on rags and burnt. Diet. Fluid diet should be given during the febrile period and until the throat is clear when this should be increased to semi solids. The bowels should be opened every other day with an enema. If the neck is swollen and painful only warm dry wool should be applied as fomentations may aid the absorption of toxin.

*Laryngeal Diphtheria* The minimum dose of antitoxin required for a child or adult is usually 20 000 to 40 000 units and much larger doses are frequently necessary. A steam kettle should be kept going day and night and hot fomentations applied to the neck. For the cough glycerin should be given undiluted in 60 doses by mouth. Tracheotomy is necessary for marked obstruction to breathing as shown by increasing restlessness dyspnoea cyanosis tachycardia and pulsus alternans. Laryngeal intubation is coming into fashion again. Laryngeal suction is also recommended. A semi soft or metal catheter is introduced by means of a direct vision laryngoscope and its distal end attached to a suction machine.

*Nasal Diphtheria* Serum must be given in large doses for nasofacial diphtheria, nasal discharge alone usually ceases after an injection of 6 000 units.

Complications requiring treatment include Serum sickness. Calamine lotion may be applied to relieve itching. Circulatory failure. The foot of the bed should be raised and pillows removed. Nothing beyond sips of water must be given by mouth and rectal injections of 4 to 8 fl oz of normal saline containing 5% dextrose should be given every 4 to 6 hours. Cardiac and respiratory stimulants such as *liq adrenal hydrochlor* m 5 *strychnin hydrochlor* gr 1/60 and *atropin sulph* gr 1/120 may be injected together hypodermically tid or Coramine (nikethramidum B P Adl) 1.5 mil tid. In peripheral

as that which produces chicken-pox, perhaps it acts through the nervous system in the former and through the blood in the latter. Its distribution is related to cutaneous nerve areas (see p. 423).

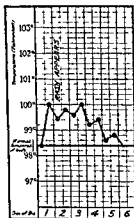


FIG. 50. TEMPERATURE CHART IN CHICKEN-POX.

**Course and Complications.** Varicella usually pursues a mild course. Complications are very infrequent, but include nephritis, laryngitis, bronchopneumonia, encephalomyelitis, conjunctivitis and otitis media. Encephalomyelitis is characterised by tremors, ataxia, or by signs of transverse myelitis. It may occur during the second week of the illness and be heralded by fever, vomiting, or a rigor. Varicella is more frequently associated with scarlet fever than with any other acute infection.

**Prognosis.** This is very good except in the severe types of the disease. Recovery usually occurs in cases of encephalomyelitis.

**Treatment.** *Prophylactic.* Inoculation with convalescent serum has not proved effective.

*Quarantine period.* This is 21 days, but the contacts need not be isolated for the first week. *Isolation period.* The patient must be isolated until the last scab has separated, which usually takes 3 to 4 weeks.

*Curative.* The patient must be put to bed until the temperature is normal, fresh spots have ceased to appear and all spots have reached the crusting stage. Usually the only treatment required, beyond that necessary for any mild feverish illness, is a local application for the irritating skin. The patient should not be allowed to scratch the spots. A dusting powder of equal parts of starch, zinc oxide and borax may be applied, and baths given when the crusts form, with warm water coloured pink with potassium permanganate. Zinc ointment (ung. zinc. ovid. B.P.) is applied to the sores left after the crusts have separated.

### Small-pox (Variola)

**Definition.** An acute infectious disease characterised by fever and a typical rash.

**Etiology.** Paschen bodies, which are present in the fluid of the pocks, are probably the causative virus. Guarnieri bodies which are found in the epithelial cells of the pocks of small-pox are considered to be agminations of Paschen bodies. Infection is spread directly, by fomites, by third persons and probably by flies, the virus being inhaled. *Predisposing causes:* 1. Absence of protection by vaccination. 2. Absence of a previous attack. 3. Season: Epidemics tend to occur in the winter and spring. 4. Race and climate: Small-pox is more common in the tropics, especially where the disease has not been endemic. 5. Age and sex: Earlier epidemics, such as those of 1783-1800 and

1837-40 affected chiefly children under 5 years now youths and adults of either sex are attacked

**Pathology** At autopsy, eruptive lesions may be seen in the œsophagus, larynx and trachea. Hæmorrhage may occur in the muscles and solid organs. Small yellow areas of focal necrosis are often found in the testicles. The spleen may be enlarged and soft.

**Incubation Period** 10 to 14 days, usually 12 days

**Clinical Findings** The patient may be a child or an adult. He is suddenly taken ill with shivering or a rigor, headache and backache

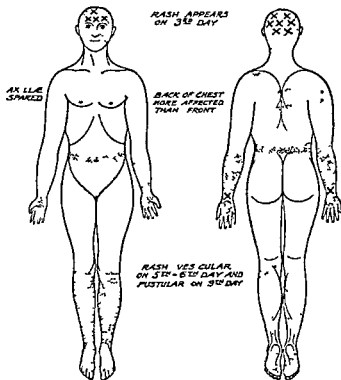


FIG 57 DIAGRAM OF RASH IN SMALL-POX

Peripheral extensor surfaces of limbs especially involved. Rash profuse on pressure points such as waist or garter line. The crosses indicate the usual site of the first appearance of the rash.

Giddiness, nausea, vomiting and severe malaise are often present.

**On Examination** During the invasion period the patient looks tired, and the temperature is often raised during the first 2 days to  $102^{\circ}$  or  $101^{\circ}$  F. Prodromal rashes may be seen, these are: 1. Petechial, involving especially the bathing drawers area (triangular rash). There are small bright or dark red spots which may also be found in the axillæ. 2. Erythematous, either scarlatiniform, morbilliform or multiform of a wider distribution. The spleen may be palpable. The true rash appears on the third day and is maculo-papular, it becomes vesicular by the fifth or sixth day, and pustular by the ninth day. The pustules burst about the twelfth day, with crust formation by the

sixteenth day. The macules first appear on the forehead, scalp and back of the wrists and spread over the face and peripheral portions of the limbs to the trunk. The rash involves especially extensor surfaces, exposed parts and those subject to pressure. The axillæ are usually spared. It is more scanty on the abdomen, loins, chest, neck and flexures of the limbs. The legs are less involved than the arms, and usually the upper half of the limbs are less involved than the lower. The eruption is also seen on the palms, soles, conjunctivæ and in the mouth (see Fig. 57).

The macules vary in size, are dark red and rapidly become papular, firm and shotty. The vesicles are pearl-coloured, and loculated so that they do not collapse on puncture. Umbilication due to depression of the centre by a sweat duct or hair follicle is often seen. The pustules

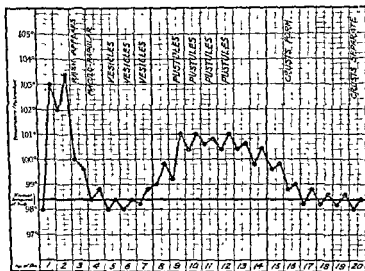


FIG. 58. TEMPERATURE CHART IN SMALL-POX.

are yellowish with a red areola and the skin around may be œdematous. The brownish-black crusts formed after rupture usually separate in from 2 to 3 weeks, leaving depressed scars which are often permanent.

There is much irritation and the skin may become tense and painful with a very offensive odour during the pustular stage. The temperature usually falls to about 99° F. or lower as the true rash appears and rises again (secondary fever) with pustulation, to about 101° F. It then gradually falls to normal about the middle of the third week (see Fig. 58). During the acute stage the patient is very prostrated and insomnia or delirium may be marked. The blood: There is leucopenia during the first 5 days and leucocytosis during the second and third weeks, with the lymphocytes forming about 40% of the count. The urine: Febrile albuminuria is often present and the diazo-reaction may be given.

*Varieties.* 1. *Discrete*: A mild type. 2. *Semi-confluent*: With



little normal skin between the spots 3 *Confluent* The pustules fuse and there is much subcutaneous oedema (These types are judged by the rash on the face and forearms and they are all varieties of *Variola vera*) 4 *Varioloid* (abortive) Slight cases, often modified owing to previous vaccination There may be no rash (*variola sine eruptione*) 5 *Variola Minor (paravariola), Alastrim and Amaas* Mild types occurring in Brazil and Africa, which have been reported in England since 1919, and were originally described in Gloucestershire by Jenner There is no secondary fever, and the rash may come out in crops Adults are chiefly affected. 6 *Hæmorrhagic* Two types are described, both very malignant (a) *Purpuric* (black small pox) with very scanty rash. Hæmorrhages occur in the skin, mucous membranes and internal organs (b) *Variola hæmorrhagica pustulosa* in which hæmorrhages occur after the development of the rash which may not reach the pustular stage Small hæmorrhages into the pocks do not constitute true hæmorrhagic small pox and are not necessarily severe 7 *Congenital*

**Differential Diagnosis** In the invasion stage small pox may be mistaken for influenza pneumonia scarlet fever, measles or purpura Search should be made for the petechial rash described above The typical features of the onset of measles and scarlet fever are absent

When the rash has appeared the differential diagnosis includes Chicken pox, measles, typhus fever, pustular drug eruptions such as those caused by iodides and bromides a pustular syphilide and acne The distribution of the rash is most important The greatest difficulty usually occurs in the case of chicken pox In small pox the rash is most profuse on the face and extremities and in chicken pox on the trunk The axilla is usually free from eruption in small pox whereas this area is affected in chicken pox In small pox the rash does not come out in crops as it does in chicken pox In small pox the typical vesicle is round and rather deep whereas it is oval and superficial in chicken pox Small pox is the only infectious disease in which the temperature falls with the appearance of the eruption

If the patient has recently been successfully vaccinated it is improbable that he is suffering from small pox If a successful test vaccination is made on the second day of illness small pox is practically excluded

**Course and Complications** The course of a case of average severity has been described above Confluent or hæmorrhagic cases are much more severe and usually rapidly fatal The complications include laryngitis bronchitis, bronchopneumonia heart failure, conjunctivitis, corneal ulceration, panophthalmitis otitis media, mastoiditis orchitis, bedsores boils encephalomyelitis, neuritis and osteomyelitis

**Prognosis** This varies with the protection afforded by previous vaccination and the type of disease The most critical days are usually the twelfth to the fourteenth If the initial fever is slight the prognosis is usually good, but the converse is not true

**Treatment** *Prophylactic* Vaccination successfully performed in infancy affords protection for about 7 years It should be repeated at the ages of 7, 14 and 21, and again if there is an epidemic Chicken pox

should be made notifiable during an epidemic of small-pox. The patient's house and its contents should be disinfected. *Quarantine period.* Contacts should be vaccinated and kept under observation for 16 days. *Isolation period.* The patient must be removed to, and kept in an isolation hospital until all the scabs have separated and the skin has healed, usually for 6 weeks or longer.

*Curative.* The patient is kept in bed in a well-ventilated room and bright light is excluded. Finsen recommended red light to prevent the rash becoming pustular, but this has not proved of value. Diet: Milk and fluids are given during the febrile period of the disease, the diet being increased when the temperature falls. Toilet: The hair should be cut short and the skin sponged twice daily with warm water. A freshly prepared saturated solution of potassium permanganate should be applied all over the skin on beginning treatment; this can be repeated in a day or so with a 1% solution. Warm baths, to which potassium permanganate is added to make it a pale pink colour, are welcomed after the pocks have burst. The virus is destroyed by 1 in 10,000 permanganate. The face should be covered with a lint mask soaked in iced water and covered with oiled silk. The offensive odour can be mitigated by dabbing the skin with 1 in 100 carbolic acid solution instead of the permanganate. The eyelids should be smeared with vaseline and the eyes frequently bathed with boracic lotion. If any eruption appears inside the lids they should be everted and treated with vaseline containing hydrarg. oxid. flav. gr. 8 and atropin. sulph. gr. 16 to the oz. daily. If there are signs of further inflammation, 10% Argyrol (argent. proteinas mite B.P.C.) should be used daily, and local heat applied frequently.

To aid the separation of the crusts, especially from the face, a thin layer of linseed poultice may be applied on a lint mask and changed every 2 hours. When the crusts have separated zinc ointment (ung. zinc. oxid. B.P.) can be applied to the scars.

To relieve the severe pain in the back and the headaches phenacetin gr. 7 or aspirin gr. 10 may be given. To induce sleep pot. brom. gr. 30, Dover's powder (pulv. ipecac. et opii B.P.) gr. 10, or paraldehyde m. 60 to 120 may be required. Delirium is best relieved by the injection of hyoscin. hydrobrom. gr. 1/100. A steam inhalation containing tnc. benzoini co. m. 60 to 1 pint relieves the laryngitis. The mouth should be cleaned after each feed with a swab dipped in glycerin. thymol. co. (B.P.C.) m. 60 to 5 oz. of water.

Heart failure may require stimulant treatment with brandy, digitalis or strophanthus. If the case is seen early, salol. gr. 10 should be given every 4 hours, as it appears to lessen the pustule formation. The bowels must be kept free with mild laxatives.

Good results are claimed from the subcutaneous injection of 25 mils of convalescent serum. This is taken from patients in the fourth or fifth week of disease, whose Wassermann reaction is negative, and who are free from tuberculosis. In a few cases sulphanilamide has been given with satisfactory results, the pustular stage not developing and the period of invalidism being shortened.

**Vaccinia**  
(*Cow-pox*)

**Definition** This is a disease of cows, but the term also includes the effects of inoculation of man with vaccine lymph containing the virus of cow pox

**Etiology** The inclusion bodies of Paschen are accepted now as the virus of vaccinia. It is probable that the virus of small pox is the same as that of cow pox, it being modified by its passage through the cow

**Vaccination** The lymph is obtained from the vesicles which result from the inoculation on the shaved abdomen of a tuberculin tested calf, of glycerinated lymph containing the virus of cow pox. The vesicular contents are mixed with glycerin and water in the proportion of lymph 20% glycerin 40% and water 40% and stored in a cool dark chamber. The lymph is not therefore standardised. Bacteriologically sterile vaccine can now be obtained by cultivating the vaccine virus in chicken's eggs. Protection against small pox was obtained by variolation or inoculation with clear lymph obtained from small pox vesicles. This was introduced in 1721 to England from Turkey by Lady Mary Wortley Montagu. It was found to be dangerous as death ensued at times or the vaccinated individual might give rise to virulent small pox in others, such variolation is now illegal. It was noted in the middle of the eighteenth century that dairy maids who had suffered from cow pox were immune to small pox. Cow pox causes vesicles and ulcers on the udder and teats of cows and is probably small pox modified by its passage through the cow. Gordon has shown that the virus of vaccinia causes vesicles on the rabbit's skin whereas that of variola will not do so. In 1796 Jenner introduced vaccination of man with cow pox lymph obtained from the hand of a dairy maid.

Vaccination is performed by placing a drop of lymph on the arm or leg and making a single linear scratch through it about a quarter of an inch long, sufficiently deep to leave a red mark but not to draw blood. Only one insertion is now recommended. The lymph is allowed to dry and may then be covered with a pad of boric lint. If a primary vaccination "takes," a papule forms on the third day, which is vesicular by the fifth day, continues to enlarge until the eighth day and matures, becoming pustular by the tenth day. The vesicle dries and forms a scab which separates between the fourteenth and twenty first days. The skin around the vesicle is red and swollen during the second week and the axillary glands are usually enlarged. A scar results which shows small depressions or foveations. During the second week there is usually malaise and some pyrexia. Primary vaccination should be done at the age of 3 months if the child is in good health. Protection is afforded against small pox for about 7 to 10 years with a primary vaccination, revaccination should be performed at the age of 7 years and again at 14 and 21 years or at any time if an epidemic occurs. Vaccination "takes" less each time and often not at all after the third time.

**Complications.** These include: 1. Sepsis: The arm may become red and brawny and very painful; the axillary glands enlarge and the temperature is raised. 2. Generalised vaccinia: This may be due to (a) auto-inoculation by scratching, the vesicles occurring elsewhere, as on the face; (b) blood-borne infection, generalised papules and vesicles forming between the fourth and tenth days. 3. Protein rashes: Various erythemata may occur. 4. Encephalomyelitis: A dread but rare complication proving fatal in about 35% of cases; it has been specially noted since 1923, and occurs usually in children and young adults who have not been previously vaccinated. It is probably due to a dormant virus activated by the vaccine virus. The symptoms are noted about the tenth or twelfth day after vaccination. The patient is drowsy, complains of headache, vomits, and becomes delirious with various pareses. Post-mortem there is perivascular demyelination in the brain and cord. Treatment consists in the intravenous or intrathecal injection of 5 to 30 mls of serum taken from an individual who has been successfully vaccinated 14 days previously.

## Mumps

### (Epidemic or Specific Parotitis)

**Definition.** An acute infectious disease characterised by enlargement of the salivary glands, usually the parotids.

**Etiology.** The virus is probably a filterable organism, present in the saliva and spread by droplets. By some it is considered to be neurotropic, the nervous system being primarily involved with secondary affection of the salivary glands. The patient is considered to be infectious for 2 or 3 days before the swelling appears. *Predisposing causes:* 1. Age: 5 to 15 years and again 18 to 25 years. 2. Sex: Chiefly males. 3. Season: Winter and spring. 4. Absence of a previous attack: Second attacks are rare. Epidemics occur all over the world.

**Pathology.** The parotid swelling is chiefly due to hyperæmia of the connective tissue. Fibrosis of the testis with atrophy of the glandular epithelium may occur. The pancreas may be hyperæmic.

**Incubation Period.** 12 to 23 days.

**Clinical Findings.** The patient is usually a boy or young adult, who complains of headache, malaise, sore throat, nose bleeding or stiff neck, before he notices the pain and swelling in one parotid region. Often, however, the parotid swelling is the first symptom.

**On Examination:** Some fulness is seen behind the angle of the jaw; this spreads forward over the masseter and down into the neck and the parotid rapidly becomes definitely enlarged. In a day or so the opposite gland is usually affected, the skin becomes tense over the gland and the jaw can only be opened slightly, owing to pain. Trismus may be noted and salivation is usually increased. More rarely the submaxillary or sublingual glands alone are affected, and here, too, there is usually a bilateral spread. When the parotid is involved the orifice of Stenson's duct in the mouth is usually seen to be swollen. Rarely the lachrymal

glands enlarge The spleen may be just palpable The temperature rises usually to  $101^{\circ}$  F or over and takes 3 or 4 days before it reaches normal With complications such as orchitis or pancreatitis it rises again The pulse is usually slow, 50 to 60 The blood There is a lymphocytosis, the average figure being 48%, and the total white cell count is increased The urine Albuminuria occurs in about one third of cases The cerebro spinal fluid An excess of lymphocytes and of globulin is frequently found, apart from meningitis

**Differential Diagnosis** This is not usually difficult If the parotid is enlarged other causes such as sepsis, drugs such as iodides, tumours and Mikulicz's disease must be excluded Septic parotitis is usually unilateral and often suppurates In Mikulicz's disease the parotid enlargement is chronic, painless and the lachrymal glands are also affected Cervical adenitis must also be excluded, such as that due to diphtheria, or glandular fever In a case of mumps in which salivary glands other than the parotid are involved, the glands are usually affected bilaterally in a day or so The lymphocytosis in mumps also aids in the diagnosis

**Course and Complications** The swelling in mumps usually disappears in less than a week and the glands practically never suppurate The most frequent complication is orchitis, which is however rare before puberty It occurs usually about the seventh day or later, generally only one testis is affected In females tenderness may occur over the ovary Other complications include acute pancreatitis as evidenced by epigastric pain, vomiting and constipation, mastitis, otitis media, labyrinthitis and nerve deafness, peripheral neuritis, and neuritis of the II, VII, VIII and III cranial nerves Encephalitis, meningism or meningitis may occur in the invasion stage or about the sixth day

**Prognosis** This is very good Permanent sterility, but rarely impotence, may result from orchitis, permanent deafness or diabetes mellitus are rare sequelae

**Treatment Prophylactic** Convalescent serum has been used to convey a temporary immunity to contacts, 15 mls being injected intramuscularly before the seventh day after exposure to infection **Quarantine period** This is 26 days, but children may attend school for the first week **Isolation period** The patient should be isolated for a week after all swelling has gone from the glands, and for a minimum of 14 days

**Curative** The patient should be in bed for 10 days, as the recumbent position lessens the liability to orchitis Fluid diet is required until mastication is painless The bowels should be opened with aperients such as salines or cascara sagrada Mouth washes should be used frequently such as glycerin thymol co (B P C.) Glycerin belladon (B P C) should be painted over the swollen glands and fomentations applied if there is much pain For orchitis the affected testicle is supported with a suspensory bandage or small pillow, and glycerin belladon (B P C) with fomentations are applied Operation is not required for pancreatitis

## Whooping-Cough

(Pertussis)

**Definition.** An acute infective disease characterised by paroxysms of coughing followed by an inspiratory whoop.

**Etiology.** Whooping-cough is most probably caused by the *Hæmophilus pertussis* (B. pertussis or the Bordet-Gengou bacillus). Some authorities believe the causative agent to be a filtrable virus, the Bordet-Gengou bacillus being a secondary infection. *Predisposing causes*: 1. Age: Chiefly children between 2 and 5 years, but adults are affected. 2. Sex: Females predominate slightly. 3. Season: March and April especially. The disease is spread by droplet infection, rarely by fomites or a third person. It is most infectious in the catarrhal stages. Second attacks may occur.

**Pathology.** Whooping-cough is rarely fatal apart from complications such as bronchopneumonia. The trachea, larynx and bronchi show catarrhal changes and the bronchial glands are enlarged. The bacillus is found especially in the larynx and trachea.

**Incubation Period.** 6 to 18 days, usually 7 days.

**Clinical Findings.** *The catarrhal stage*: The patient is generally a child, who first shows the symptoms of a severe cold and a little cough. The temperature is slightly raised, 99° to 100° F. The blood: There is a leucocytosis of 12,000 to 27,000 per c.mm, with 60% or more lymphocytes. *If the patient coughs over a blood-agar potato plate the Bordet-Gengou bacillus can often be isolated.* *The paroxysmal stage*: This begins about 5 to 14 days later. It is characterised by a noisy, rapidly repeated, explosive cough, during which the child is cyanosed, tears may run from the eyes, and mucus from the nose. This is immediately followed by a long-drawn crowing inspiration or whoop. The attacks tend to be provoked by feeding, by exertion or emotions, but they also occur at night. After the attack the child may drop asleep. She is often alarmed by the paroxysm and may get out of bed or run to her mother. Vomiting may occur at the end of the attack and the patient lose weight. In the intervals the conjunctivæ tend to be congested, and the face a little swollen, ulceration of the frænum linguæ may be seen, due to friction against the teeth during the attacks. The temperature is usually normal. The blood still shows a leucocytosis. The urine may contain an excess of uric acid.

In some cases the attack is mild or abortive, and although paroxysms of barking cough occur by day and night, there is no whoop. *The convalescent stage*: After about 4 to 6 weeks the whoop disappears, and the cough gradually lessens, although it may still be provoked by running or sea-bathing. The leucocyte count has now returned to normal.

**Differential Diagnosis.** Whooping-cough must be diagnosed from other catarrhal infections such as a cold, the early stages of measles and laryngeal diphtheria, from paroxysmal coughs due to tracheo-bronchitis or enlarged bronchial glands, which in children are often tuberculous, and from other causes of mediastinal pressure in adults.

If there is no whoop the diagnosis depends upon the character of the cough the blood count and isolation of the bacillus from droplets or sputum. A complement fixation test and an intradermal test (using Sauer's vaccine made from strongly hemolytic strains of the *Hæmophilus pertussis*) have also been used for the diagnosis of whooping cough. The CFT becomes positive early in the infection in a considerable proportion of cases. The results with the intradermal test are not so reliable.

**Course and Complications** The usual course is as described above. The most important complications are Bronchitis, bronchopneumonia, encephalitis, convulsions and cerebral hæmorrhage. Other complications include spasm of the glottis, bronchiolectasis, acute emphysema, surgical emphysema, spontaneous pneumothorax, otitis media, prolonged vomiting, bleeding from the nose, ears, eyes and gums. The cough may provoke hernia or prolapse of the rectum. Children in hospitals and school sanatoria are very liable to contract diphtheria and a prophylactic injection of 1 000 to 2 000 units of diphtheria antitoxin is advisable. Bronchial gland tuberculosis may supervene.

**Prognosis** The disease is serious in very young infants, in debilitated and rickety children and in association with convulsions and bronchopneumonia. In an uncomplicated case the severity may be gauged by the number of paroxysms; any figure over 20 in the 24 hours being grave. The outlook is bad where there is inability to retain food, or when there are frequent convulsions.

**Treatment** *Prophylactic* Infants and children up to age of 8 years should be immunised with H. pertussis vaccine made from killed Phase I strains. The first injection is best given about the age of 12 months. The dosage is 4 000 millions repeated after a week, again after a month and again a month later. A further injection should be given a year later, or when the child is first exposed to whooping cough. The intramuscular injection of 10 mls of convalescent serum confers immunity for about 3 weeks and this method should be used for the protection of unvaccinated contacts. *Quarantine period* This is 3 weeks. *Isolation period* This is 6 weeks.

**Curative** The patient should be in bed during the catarrhal stage and until the vomiting becomes infrequent. The room should be well ventilated but maintained at a temperature of 60° F. Good results have been obtained by treating patients in the open air, suitably clothed and kept warm in bed with hot water bottles. The children are less irritable. **Diet** Liquids and semi solids are advisable until the paroxysms lessen. When vomiting is severe the feeds should be given about 10 minutes after the vomit as this offers the best chance of the nourishment being retained. Dextrose gr 60 should be given t.i.d. to prevent acidosis. Discharges and sputum should be burnt. An inhalation of the benzoin co m 60 to 1 pint of steaming water often relieves catarrh. For the cough a sedative mixture containing Tnc. opii camph m 5, tnc belladon m 7, syr pruni serot. m 10, aq chlorof ad m 120 should be given six hourly for a child of 5, and the belladonna may be increased until the pupils dilate. Sleep may be

aided by Sedobrol tablets,  $\frac{1}{2}$  to 1 in half a cup of hot water for a child of 5. If the paroxysms still continue a rectal injection of ether m. 60 and olive oil fl. oz.  $\frac{1}{2}$  may be given twice daily, or phenobarbitone, gr.  $1\frac{1}{2}$ , t.i.d. in milk for an infant. Vaccine treatment is of little value and may make the patient worse. Treatment by injections of convalescent serum, 20 to 40 mls, has been tried without great success. Intramuscular injections of 2 mls of ether are not advised owing to pain. Convulsions in an infant should be treated with hot baths, lumbar puncture, inhalations of chloroform, or the rectal injection of pot. brom. gr. 6 in water fl. oz. 1. Sulphanilamide should be given for 5 days to minimise the risk of bronchopneumonia and otitis media (see p. 528).

General ultra-violet light radiation is useful. Subsequently, during convalescence, a mixture containing equal parts of cod-liver oil and malt, or Parrish's food (syr. ferri phosphat. co. B.P.) should be given in doses of m. 60 t.i.d. for a child of 5.

### Typhoid Fever (Enteric Fever)

**Definition.** An acute infective disease characterised by continued fever, enlargement of the spleen, bacillæmia, involvement of intestinal lymphatic tissue and usually a roseolar eruption.

**Etiology.** Typhoid fever is caused by the *Bacterium typhosum* (B. typhosus), of which 7 strains have been identified. **Predisposing causes:** 1. Age: Chiefly between 5 and 35 years. 2. Sex: Slight excess of males. 3. Season: Chiefly the autumn. 4. Overcrowding: Especially in tents in the army. 5. Bad sanitation. 6. Absence of a previous attack or recent inoculation. The disease occurs all over the world. It is spread by patients and carriers, through contamination of food and water, especially milk, cream, butter, oysters, ice-cream and water-cress; it is also spread by flies (their feet, vomit and excreta), by fomites, and by inhalation of bacilli, as from dried excreta. The nurse in charge of a patient may contract typhoid fever by the latter method. Epidemics are usually due to a carrier contaminating the food supply of an area, owing to bacilli on his fingers soiled with urine or feces, or to sewage contamination of drinking water.

**Pathology.** Infection probably spreads from the bowel to the blood; the organisms are then excreted by the bowel or kidneys. The bacilli tend to lodge in the gall-bladder and intestinal lymphoid tissue, and they may be found in the cutaneous spots. Intestinal lesions include swelling of Peyer's patches and solitary lymphatic follicles; the last 18 inches of the ileum are chiefly involved. These areas may ulcerate and perforate. The spleen is enlarged and soft, the mesenteric glands are enlarged, the skeletal muscles may undergo Zencker's degeneration.

**Incubation Period.** 7 to 21 days, usually 14 days.

**Clinical Findings.** The onset is usually insidious, the patient complaining of lassitude, frontal headache, backache, constipation, anorexia, epistaxis, malaise and insomnia associated with a gradually



rising temperature In some cases there is a sudden onset with fever, vomiting rigors and delirium As the disease runs an average course of 4 weeks the main features during each week will be considered

**First Week** (Invasion stage or advance) The patient may be up (ambulatory typhoid) the face is a little flushed and the tongue has a white fur with the edges and tip clean The pupils are dilated There may be complaint of abdominal pains or discomfort The temperature rises in a step ladder fashion with a progressive evening rise and it drops  $\frac{1}{2}$  to 1 degree each morning In this way by the end of the week it may reach  $102^{\circ}$  to  $103^{\circ}$  F (see Fig 59) The pulse is relatively slow, 90 to 100, and often dicrotic The abdomen is a little swollen owing to flatulence, and there may be gurgling on palpation over the cæcum The abdominal reflex is usually absent The spleen is just palpable Typhoid spots (rose spots) may appear about the

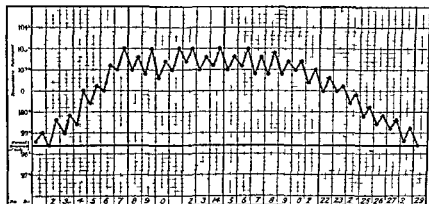


FIG 59 TEMPERATURE CHART IN TYPHOID FEVER

seventh day pink macules 2 to 4 mm in diameter fading on pressure and situated on the trunk and abdomen They are not numerous perhaps 6 to 12 they disappear in 2 to 4 days and fresh crops appear There is often a little bronchitis The blood There is leucopenia 4 000 to 5 000 white cells per c mm At times times there is a slight leucocytosis during the first week Typhoid bacilli are present The urine Febrile albuminuria may occur The diazo reaction is positive Typhoid bacilli may be present at the end of the week but more often not until the second week The motions may be costive or loose ( pea soup ) yellowish and offensive Typhoid bacilli may be present but they are found in a higher percentage of cases in the second and third weeks During this time there may be swelling of Peyer's patches

**Second Week** (Fastigium) The patient is more prostrated the headache is less marked and deafness may develop The tongue is drier and coated in the centre with a clean tip and edges Insomnia may now be troublesome and delirium occur The temperature remains sustained at about  $101^{\circ}$  to  $103^{\circ}$  F and the pulse accelerates a little to just over 100 The blood The Widal reaction becomes positive after the tenth day, typhoid bacilli are usually present The bowels There

is a greater tendency to diarrhoea during this period. During this week sloughs may form in Peyer's patches.

*The Third Week. (Defervescence).* The patient is still more exhausted and may sink into the "typhoid-state" with delirium, muscular twitching, coma-vigil, a dry and shiny tongue and sordes on the lips. It is the danger period, owing to the risk of hæmorrhage or perforation. During this week the intestinal sloughs may separate. Usually there is, however, improvement towards the end of this week and the temperature begins to fall by lysis; rarely by crisis. The abdomen may be more distended by meteorism, and the patient is often much wasted.

*The Fourth Week. (Convalescence).* The temperature gradually falls to normal in the mornings, rising a little in the evenings. The abdominal reflex reappears, the spleen is no longer palpable, and the general condition improves. During this week repair takes place in the areas where the sloughs have separated. Recrudescences may occur, the temperature rising irregularly, but the spleen does not enlarge and no fresh spots appear. *Relapses:* After the temperature has been normal for about a week, it may rise again in step-ladder form and the disease be repeated, with fresh spots, enlargement of the spleen and intestinal symptoms. Relapses are more common after a high calorie diet has been given. The duration of the relapse is usually shorter than that of the original disease. *Varieties:* 1. *Mild* (including "ambulant" cases). 2. *Pyrexial*. 3. *Severe*. (a) *Hæmorrhagic*. (b) *Pneumotyphoid*, the disease starts with lobar consolidation of the lung. (c) *Nephro-typhoid*, the onset is with symptoms of acute nephritis. (d) *Meningo-typhoid*, the onset is with symptoms of meningitis.

*Differential Diagnosis.* At the onset typhoid fever may be mistaken for influenza, gastro-enteritis, pneumonia, nephritis or meningitis. The temperature should always be taken, as ambulant cases will then be kept under observation when found to be pyrexial. The continued fever may be confused with that due to paratyphoid fever, tuberculous peritonitis, miliary tuberculosis, pyæmia, undulant fever, infective endocarditis, or Hodgkin's disease. Typhus fever and secondary syphilis have also caused errors of diagnosis. A typical clinical picture is not always present, and reliance must be placed on blood and siccæ culture and the Widal test. A person inoculated with T.A.B. vaccine will give a positive Widal reaction for a year or more, but if an active typhoid infection supervenes the agglutinin titre of the serum usually waxes and wanes during the course of the disease. Virulent strains of typhoid bacilli produce two varieties of antigens, O. and Vi.; the third antigen, H., is not an indication of active infection. These antigens give rise to corresponding agglutinins in the serum. Agglutination of the H. form is probably indicative of previous inoculation, whereas O. agglutination signifies active infection.

*Course and Complications.* The typical course has been described above, together with the recrudescences and relapses which ensue at times. The most important complications are:—Intestinal: (a) Hæmorrhage. Especially during the third week. The temperature falls, the

pulse becomes rapid and feeble, there is pallor, and air hunger may ensue. Blood is seen in the motions in varying amounts. (b) Perforation and peritonitis. This is likely to occur at the end of the third week. There is abdominal pain, which may be referred to the tip of the penis, owing to the contiguity of the bladder. The patient's expression becomes drawn and anxious. The effect on the temperature varies, it may or may not fall. The pulse is more frequent. There is some limitation of abdominal movement with respiration, the abdomen may be a little more swollen, tenderness and rigidity may be present and the patient usually lies with the legs drawn up. Venous thrombosis. The left femoral vein is usually affected, often in the fourth week. Respiratory. (a) Laryngitis or ulceration of laryngeal cartilages. (b) Bronchitis. (c) Pneumonia in the third week or later, or at the onset (pneumo-typhoid). (d) Pleural effusions. Cardiac. Myocardial degeneration. Endocarditis and pericarditis are rare. Neuritis, especially causing the "tender toes".

Other complications include Otitis and parotitis, which are not rare, meningitis, cerebral thrombosis or embolus, myelitis, nephritis, typhoid spine (thickening of the intervertebral discs, it usually occurs late in the disease and the pain is very severe), periostitis, boils, acute cholecystitis, infarction of the spleen and suppuration of the mesenteric glands. "White leg" following venous thrombosis, or gall stones may result as sequelæ of typhoid fever.

**Prognosis.** Typhoid fever is a serious disease, the gravity varying largely with the extent and depth of intestinal ulceration. A high temperature and a pulse over 110 are unfavourable signs.

**Treatment.** *Prophylactic.* The disease is preventable. Contacts and all exposed to infection should be inoculated. Inoculation does not increase the risk of infection. A mixed vaccine (T.A.B.) containing 1,000 millions typhoid and 500 millions paratyphoid A and B in 1 ml., is used. 0.5 ml. is first given and after 10 days 1 ml. is injected subcutaneously. This vaccine may be combined with 1 ml. of tetanus toxoid in each dose and repeated at an interval of 1 month. Pulmonary tuberculosis is a contra-indication. Vaccines administered by mouth are not so effective. The immunity conferred lasts about 2 years. In an epidemic, water and milk should be boiled and carriers sought for. There is no medical treatment which will inevitably cure a chronic carrier. In some cases cholecystectomy has been successful. All cases must be notified. *Isolation period.* The patient must be isolated for about 5 to 6 weeks and on discharge the fæces and urine should be free from typhoid bacilli on two successive examinations at an interval of 1 week. Should the patient continue to excrete typhoid bacilli after 3 months he becomes a carrier. A positive Vi agglutination does not indicate that the individual is a carrier.

**Curative.** The patient is put to bed and kept there until the temperature has been normal for 2 weeks. He must at first be kept lying flat, and turned frequently from side to side to prevent bed sores, air rings should be used under the pelvis and other pressure points, such as the elbows and heels. The back must be treated daily with spirit and

powder, and the body sponged twice daily with tepid water all over. The nurse should wear gloves while attending to the patient and wash her hands well in soap and water, and afterwards rinse them in 1/2,000 perchloride of mercury. Diet: Patients undoubtedly do better when given an adequate dietary of 2,000 to 3,000 calories, which prevents tissue waste. Convalescence is usually thereby shortened, the mortality lowered, and the incidence of hæmorrhages and perforation is much reduced. Too much milk should not be given as the curds formed are irritating to the intestines. Sodium citrate gr. 2 to the ounce of milk may be added, if there is curd indigestion. The patient should drink 4 to 6 pints of water or lemonade daily, in addition to his food. Part of this fluid can be replaced by dextrose-lemonade, containing  $\frac{1}{2}$  lb. of dextrose in 1 pint of water, flavoured with the juice of 2 lemons. Feeds should be given regularly every 2 hours while the patient is awake. The following diet can be given, with a value of about 2,800 calories.

- 7 a.m. Milk 4 oz., water 2 oz., cream  $\frac{1}{2}$  oz., lactose  $\frac{1}{2}$  oz., 1 rusk and  $\frac{1}{2}$  oz. butter.
- 9 a.m. Beef tea, well salted, 1 cup.
- 11 a.m. Milk 4 oz., water 2 oz., cream  $\frac{1}{2}$  oz., lactose  $\frac{1}{2}$  oz.
- 1 p.m. Mashed potato with butter, 2 tablespoonfuls.
- \* 3 p.m. Lightly boiled or poached egg, with rusk or toast and butter ( $\frac{1}{2}$  oz.).
- 5 p.m. Cornflour or well-boiled rice or egg custard, 5 oz., and cream  $1\frac{1}{2}$  oz.
- 7 p.m. Beef tea, 1 cup.
- 10 p.m. Milk 4 oz., water 2 oz., cream  $\frac{1}{2}$  oz., lactose  $\frac{1}{2}$  oz., 1 rusk or toast and  $\frac{1}{2}$  oz. butter.

Two night feeds of milk 4 oz., water 2 oz., cream  $\frac{1}{2}$  oz., lactose  $\frac{1}{2}$  oz. The milk feeds can be flavoured with tea, coffee, cocoa or chocolate. Further additions can be made to the diet when the morning temperature falls to normal. These include sponge cakes, pounded fish, minced meat and thin bread and butter. The mouth should be cleansed after each feed with a swab dipped in glycerin and lemon. The bowels: The stools should be examined daily for undigested food, or blood or membrane. If there is constipation an enema may be given on alternate days, or liq. magnes. bicarb. fl. oz. 1 by mouth. If there is diarrhœa, meat extracts and cream should be omitted and the milk should be peptonised. If the diarrhœa still persists a starch and opium enema (starch gr. 60, water fl. oz. 2 and tnc. opii m. 30) should be administered. Meteorism is likely to occur if too much milk is given. For its relief turpentine stupes can be applied to the abdomen, the flannels being wrung out in water containing ol. terebinthin. m. 60 in 2 pints of water. If the temperature rises above 104° F. it should be lowered 2 degrees by tepid sponging. Drugs: Oil of cinnamon m. 3 to 5 in capsules may be given two-hourly. The use of "anti-Vi" and "anti-O.", serum (Felix), 25 mls, injected intramuscularly, and in severe cases repeated in 24 hours, is still in the experimental stage, and the injection

cows or pigs. Man may be infected by drinking contaminated cow's milk, or by handling infected material from a cow or pig. The causative organism is closely related to that producing Mediterranean fever (see p. 708). Abortus fever has assumed a greater importance owing to the number of cases which have been recorded in America, England and in other European countries. Eighty per cent. of milch herds in England are infected with the bacillus abortus, and the majority of the infected animals excrete the organism in their milk at some time. At least 25% of the unpasteurised milk supply to the large towns in England contains the Br. abortus. Cream is a less common source of infection, and butter and cheese are doubtful sources. *Predisposing causes*: Sex: Males predominate slightly. Age: 15 to 45.

*Pathology.* A septicæmic infection occurs. The organisms are excreted in the urine. Post-mortem the spleen and liver may be found enlarged.

*Incubation Period.* 5 to 15 days.

*Clinical Findings.* The disease is usually milder than Malta fever. The patient is gradually taken ill with malaise, headache and pain in the back and limbs. The onset may be sudden with a rigor. Pains may also be felt in the jaws, eyeballs or testicles. In some cases the throat is sore and swallowing is painful.

*On Examination*: The most striking feature is the continued fever. The temperature usually rises irregularly and remains remittent for 1 to 4 weeks. After falling to normal, the temperature may rise again in one or two days, but the second wave is generally shorter than the first. The temperature may then settle to normal with an occasional rise to 99° F., or for a prolonged period, such as 8 months, irregular rises may occur. The pulse is usually slow, about 80. There is often marked sweating in the early morning, and transient painful swellings may occur in the joints. The bowels tend to be constipated. The spleen is palpable in some cases. Less frequently the liver and lymph glands may be enlarged. Epistaxis may occur late in the disease. A vesicular eruption may be seen on the palate. The blood: There is leucopenia with a relative lymphocytosis of about 50%, and some anaemia. The agglutination test is usually first positive between the sixth and fourteenth days. An agglutination in a dilution of 1/80 is considered to be diagnostic. Often agglutination in a dilution of 1/1,000 or more is obtained. The blood culture is often positive, but the organism may not grow for 19 days or longer, under anaerobic conditions. *Varieties*: Mild attacks probably frequently pass unnoticed. A severe septicæmic form is also described.

*Differential Diagnosis.* The disease must be distinguished from other causes of prolonged fever, sweating, and joint pains, such as enterica infections, tuberculosis, malaria, septic infections, rheumatic fever and influenza. The diagnosis rests upon the agglutination test, the leucopenia, and the isolation of the organism from the blood. There is also a melitene reaction, performed by injecting intradermally 1/20 mil. of the filtrate of a broth culture of the organism. A positive result is shown by the formation of a red swelling which persists for nearly

two days This reaction is usually better marked with melitensis than with abortus infections

**Course and Complications** As described above, the course is characterised by a tendency to relapse, and so it is difficult to say when the disease is cured Complications include bronchitis orchitis abortion, mastitis, proctitis, arthritis and pains in the extremities

**Prognosis** The mortality rate is 2 to 3%, and the disease is often serious owing to the tendency to prolonged invalidism

**Treatment Prophylactic** Only pasteurised or boiled milk should be drunk The treatment of infected cows is still undecided Living vaccines are usually administered to check the abortion, but there is a risk that the cows will then become chronic carriers, eliminating the organisms in their milk

**Curative** The patient should be put to bed and given an initial dose of calomel gr 3 with mag sulph gr 60 next morning The diet should be liquid during the pyrexial waves, such as boiled or pasteurised milk, dextrose orangade, barley water and meat extracts but nourishment should be increased during the apyrexial intervals Pains may be relieved by aspirin gr 10 t d s, or phenacetin gr 5, as required If the temperature is above 103° F, it should be lowered two degrees by tepid sponging The results of sulphonamide therapy are not very good About one half to one quarter of the cases show definite benefit after the administration of sulphanilamide or Sulphapyridine (M & B 693) The former appears more efficacious The dose for an adult is 1 G t d s for 5 to 7 days A second course should be given if there is a relapse If the fever continues to relapse the question of an intra venous injection of 50 to 100 millions of T.A.B vaccine should be considered This appears to cut short the fever by protein shock, the temperature shooting up to 105° F or so with a rigor and considerable signs of collapse In a favourable case the temperature falls to normal within 30 hours This treatment should not be used in the elderly, or if there are signs of myocardial weakness The urine and faeces should be disinfected as for typhoid fever (see p 559)

### Typhus Fever

#### (Spotted Fever)

**Definition** An acute infectious disease characterised by fever, a typical rash profound toxæmia and nervous symptoms

**Etiology** Typhus fever is believed to be caused by Rickettsia prowazeki bodies oval filter passing organisms These are found in the patient (internal organs) and in lice (pediculi corporis and also capitis) which transmit the disease Typhus fever is spread by lice, either by their bites or by their excreta being scratched into the skin The louse does not become infective for 6 to 7 days after biting a patient with typhus Flea borne and tick borne forms of typhus also occur **Predisposing causes** 1 Under nutrition and famine 2 Dirt and overcrowding 3 Season Winter and spring 4 Age and sex All ages and both sexes are affected 5 Locality It is more common in Eastern than in Western Europe It occurs in North and Central Asia North

Africa, and near the Andes in South America. It is unknown in the tropics except at great altitudes. It occurs endemically and in epidemics. A mild form is called Brill's disease in America and Tabardillo in Mexico.

**Pathology.** Typhus nodules are found microscopically post-mortem in the skin, brain and other organs. They are due to localised necrosis and dilatation of small vessels. The spleen is enlarged and soft, and petechiæ may be seen in various mucous membranes.

**Incubation Period.** 5 to 21 days, usually 12 to 14 days.

**Clinical Findings.** There may be a prodromal period, with malaise, for a few days. The patient is usually suddenly taken ill with frontal or occipital headache, pains in the back and limbs, shivering, nausea, or rarely vomiting.

**On Examination:** The patient is flushed and looks rather drowsy; the eyes are congested and the tongue coated. The temperature is raised to about 100° F.

The rash appears on the fourth or fifth day, first as a "subcuticular mottling," the spots being under the skin, and later papules develop. These are rose-pink, fading on pressure and resemble typhoid spots. They enlarge, become darker, brownish and do not fade on pressure. Small petechiæ may be seen between the spots. The whole rash is called a "mulberry" rash. It appears first on the trunk and spreads to the limbs, the face is usually exempt, but is red and swollen. The rash fades during the second week and leaves some staining. It does not appear in crops. The skin emits a peculiar mousy odour. The temperature: This usually rises irregularly during the first 4 days to about 103° F., there is then a continued fever for about a week, when the crisis occurs about the tenth or fourteenth day. The fall is critical but may take 2 to 3 days before normal is reached. The pulse increases in rate with the rise in temperature. The respirations are usually increased to about 20 per minute. The blood: There is a leucocytosis, with excess of monocytes (up to 15%). A special agglutination is given by the serum, called the Weil-Felix reaction. The serum agglutinates, in the later stages of the disease, in high dilutions (over 1/1,000) cultures of a proteus-like bacillus obtained from the urine of typhus patients, called the OX19 bacillus. This reaction is characteristic of typhus and appears about the seventh day. A marked agglutination by the macroscopic method of 1/100 is considered positive at this stage. The Wassermann reaction is usually positive before the crisis. The urine: Febrile albuminuria and a positive diazo-reaction are present. The cerebro-spinal fluid is clear, but may contain an excess of globulin and leucocytes.

The general condition throughout the febrile period is one of extreme prostration; an excited delirium may occur during the first week, followed by "coma-vigil" in the second week, in which the patient lies semi-conscious with eyes partially open and muscles twitching. The patient may be tortured by fearful delusions. The pupils are usually small and the knee-jerks are absent, the tongue becomes dry, black and cracked. The bowels are often constipated early and diarrhoea

may occur in the second week. Thirst is marked and there may be some deafness, the spleen is usually just palpable. In a favourable case when the temperature falls the condition rapidly improves.

**Varieties** 1 *Mild typhus* 2 *Severe or blasting typhus* (*Typhus siderans*) which is rapidly fatal 3 *Typhus sine eruptione* may occur, in which the Weil-Felix test is positive.

**Differential Diagnosis** Typhus is very rare in England, and when an isolated case occurs it is most likely to be mistaken for typhoid fever or measles. Other conditions which may require exclusion are Influenza, cerebro-spinal fever, malignant malaria, encephalitis, purpura, central pneumonia, relapsing fever or uraemia. The marked prostration, typical rash and Weil-Felix reaction serve to differentiate typhus.

**Course and Complications** A relapse is extremely rare, but a second attack may occur. Complications include Laryngitis, Bronchitis, myocardial degeneration, venous thrombosis, otitis media and nephritis. Permanent mental deterioration, inability to concentrate or headaches may be sequelae.

**Prognosis** Typhus fever is a serious disease and the mortality increases with the age of the patient. Practically all patients over the age of 50 die. The death rate is low in the endemic form.

**Treatment** *Prophylactic* Freedom from lice is all important. A short-lived immunity can be conferred on contacts by subcutaneous injection of 10 mls of serum taken from a convalescent whose temperature has been normal for 2 weeks. Many attempts have been made to obtain active immunisation by injection of rickettsiae living or killed. A vaccine prepared by formalising suspensions of rickettsiae grown in mouse lings has met with some success. *Quarantine period* Contacts should be deloused and quarantined for 15 days. *Isolation period* The patient should be isolated for 5 weeks.

**Cure** The patient should be taken to an isolation hospital, his clothes removed, the axillary and pubic hair shaved, the head closely clipped and treated with sassafras lotion (ol. sassafras (B.P.C.) fl. oz.  $\frac{1}{2}$  ol. amygdal. m. 60) with a stiff brush to destroy nits and bathed in soap and water. The attendants should have their hair and persons protected from lice by caps and overalls and they should wear rubber gloves. The patient is put to bed in an airy room. Plenty of fluids should be given by mouth to aid toxin elimination. The skin should be tepid sponged twice or thrice a day. The mouth should be cleaned after feeds with glycerin and borax or glycerin thymol co (B.P.C.). The bowels should be opened as required with an enema, if there is retention of urine, catheterisation at eight hourly intervals must be performed. For the excited delirium sod. phenobarbiton gr. 1 and sod. brom. gr. 30 or paraldehyde m. 60 to 120 may be given by mouth or it may be necessary to inject subcutaneously hyoscine hydrobrom. gr.  $\frac{1}{200}$ . If there are signs of collapse cardiac stimulants such as hot drinks, whisky fl. oz. 2 in 24 hours or the subcutaneous injection of digitalin (gr.  $\frac{1}{100}$ ) or Coramine (nikethamidum B.P. Add.) 1.5 ml. may be given four hourly. Treatment by injection of convalescent serum has not



yielded striking results, but Glutard has obtained good results by intravenous injection of hexamine gr. 22 in 5 mls of water, increased to gr. 44 daily for 1 to 3 days, until the temperature falls. He claims to have halved the mortality of the disease by this treatment.

### Erysipelas

(*St. Anthony's Fire*)

**Definition.** An acute infective disease characterised by fever and a typical inflammation of the skin.

**Etiology.** Erysipelas is due to infection with a special strain of hæmolytic streptococcus (*streptococcus erysipelatis*). *Predisposing causes:* 1. Alcoholism. 2. General debility and defective sanitation. 3. Season: Autumn and winter. 4. A previous attack: Recurrences are very common. 5. Age: It occurs in new-born infants and subsequently chiefly between the ages of 20 and 60. 6. Sex: Slightly more common in females.

The organisms enter the skin through a minute abrasion or through operation wounds or birth injuries (puerperal erysipelas). They are conveyed by direct contact or by contaminated hands or instruments, or the infection may occur apparently independently.

**Pathology.** The organisms are found chiefly in the lymphatics at the edges of the inflamed area of skin, and spread to the subcutaneous tissues may cause suppuration. Toxæmia is often severe.

**Incubation Period.** 2 to 3 days or longer.

**Clinical Findings.** The patient, who is usually an adult, may give a history of some trivial injury to the face, or may be recovering from an operation or childbirth. There is usually a sudden onset with shivering, headache, malaise, and at times vomiting and a sore throat. A burning sensation is then felt on a certain area of the skin.

**On Examination:** A bright red patch is seen, with a definite raised margin. This spreads and the skin becomes tense and shiny, and vesicles or blebs containing yellowish fluid may form on the red area. This fluid usually does not contain streptococci.

Facial erysipelas usually begins near the nose or inner canthus of the eye. The face may become very bloated and the eyelids so œdematous that the patient cannot see; the ears also become red and tense, and the tongue is very furred. The tissues are at this stage very painful on pressure, and the skin feels tightly stretched. The glands in the neck are enlarged. Headache, insomnia and noisy delirium may be troublesome.

Erysipelas may spread from one part of the skin to another (wandering erysipelas), or the lesion may first appear at such sites as the back, around the umbilicus in newborn infants, around the vulva during the puerperium, and around any operation wound which is usually a "clean" one. The fauces may be red and swollen (faucial erysipelas) or the mucous membrane of the nose affected. The temperature rises rapidly to 103° or 104° F., and remains irregularly raised for about 6 to 8 days; it often falls by crisis or may gradually subside. The pulse is proportionately frequent. The blood shows a polymorphonuclear

leucocytosis The urine usually contains albumin When the rash fades it is followed by desquamation

**Differential Diagnosis** Erysipelas must be diagnosed from simple inflammations In the latter there is not a raised, red edge, and the central part of the inflamed area is more angry looking than the periphery The constitutional disturbance is not as great

**Course and Complications** The usual course is as described above, but mild, severe or protracted attacks occur Relapse may develop after the temperature has been normal for a day or so Complications include septicæmia, subcutaneous abscesses and bronchopneumonia

**Prognosis** Erysipelas is frequently mild, but the outlook is grave in an alcoholic, in the newborn and in the aged

**Treatment.** The patient should be kept in bed and isolated until the temperature is normal and the lesions healed The diet should be liquid or semi solid while the temperature is raised Plenty of fluids should be drunk, and the bowels opened daily with salines or enemata Paraldehyde fl oz  $\frac{1}{4}$  to  $\frac{3}{4}$  may be given to an adult for insomnia Noisy delirium may be checked with an injection of hyoscin hydrobrom gr 1/200 to 1/100 For headache, an ice bag may be applied Cardiac and respiratory stimulants are necessary in severe cases, such as Coramine (nikethamidum B P Add) 15 mil., digitalin gr 1/100 strychnin hydrochlor gr 1/60 or liq adrenalin hydrochlor m 5 subcutaneously four hourly Brandy or whisky fl oz  $\frac{1}{2}$  six hourly should be given to alcoholics

**Local Treatment** The affected skin may be covered with lint wrung out in an iced saturated solution of magnesium sulphate and covered with oiled silk Good results have also been obtained by the use of ultra violet light The eyes should be washed out with boric lotion and a drop of 5% Argrol (argent proteinas mite B P C) instilled if they become inflamed

Specific treatment has not proved of great value, vaccines are not helpful, 40 mls of concentrated scarlet fever streptococcus antitoxin may be given intramuscularly into the outer side of the thigh, or 10 mls of anti erysipelas serum (erysipelas antitoxin) repeated in 24 hours The mortality has been lowered by the use of sulphanilamide The average dose is 8 G during the first 24 hours (2 G, 2 G, 1 G, 1 G, 1 G and 1 G, 4 hourly), and 6 G (1 G 4 hourly) every 24 hours during the next 8 days, given in tablet form by mouth

## CHAPTER VIII

### INFECTIOUS DISEASES OF KNOWN AND DOUBTFUL ETIOLOGY

#### Syphilis (Lues)

**Definition.** A venereal and general disease caused by a specific protozoon.

**Etiology.** The causative agent is the *Treponema pallidum* (*Spirochæte pallida*). Infection is usually by direct transmission in sexual intercourse. The primary sore and secondary lesions, such as condylomata, are very infectious, and the treponemes may also be present in the saliva and urine in the secondary stage. Localised gummata, such as may occur in the throat, are also sources of infection. Syphilis less often results from other causes, as by contact with an infected article such as a cup or a pipe, or the primary lesion may occur on a doctor's examining finger (*syphilis innocens*). The disease may be transmitted by an infected mother to her offspring (congenital syphilis). The organisms usually enter through an abrasion in a mucous membrane.

**Pathology.** The initial lesion is the primary sore or chancre. The treponemes pass to the neighbouring lymphatic glands and rapid dissemination by the blood stream ensues, probably as soon as, or even before, the chancre is visible. The primary sore is usually genital, but extragenital chancres may occur on the lip, tonsil, tongue, breast, finger and elsewhere. The secondary stage is the clinical manifestation of generalisation of infection; lymphatic glands are enlarged, especially the posterior cervical, axillary and epitrochlear. Various rashes occur due to reactions around cutaneous blood vessels. The kidneys may show evidence of nephrosis. Gumma formation is the characteristic feature of the tertiary stage, the gumma being a granuloma which is comparatively avascular. It may occur in the skin, the heart or lungs, the liver, the central nervous system, bones, and in other tissues. The arteries are especially affected in syphilis, there being early, a round-celled infiltration of the adventitia. The inflammatory change spreads inwards along the vasa vasorum to the media, where rupture of elastic fibres occurs. In the aorta this predisposes to aneurysm. The mouths of the coronary arteries are constricted, but the vessels themselves are unaffected. Endarteritis obliterans may occur in the smaller vessels. Treponemes are most numerous in the primary sore and the neighbouring glands; they occur in the blood, cerebro-spinal fluid, and in the cutaneous lesions; they are most scanty in the tertiary lesions, but lie dormant there, being sheltered from blood-borne spirochætocidal drugs, owing to the interferences with the blood supply.

### Congenital Syphilis

**Clinical Findings** A history is often obtained that the mother has had a series of miscarriages or still births before a living child is born. At birth the infant may be normal in appearance, or small. The skin may be sallow (*café au lait*), the face old looking, and the cry rather squeaking. A bullous or pustular eruption may be present at birth. The various manifestations of congenital syphilis are best classified under the age periods at which they are likely to appear.

**At Birth** Much hair on the head (syphilitic mop). Aged appearance. Bullous rash (syphilitic pemphigus), especially on the palms and soles. White pneumonia (due to fibrosis of the lungs).

**Three to Four Weeks** Syphilitic roseola, especially around the buttocks, "snuffles" due to rhinitis, otitis media. Choroiditis and iritis. Paroxysmal hemoglobinuria.

**Three to Four Months** Epiphysitis, causing apparent paralysis of limbs (pseudo-paresis). Rhagades (fissures) at the angles of the mouth, which leave radiating scars on healing. Condylomata in the perineum or under the arms. Enlargement of the spleen and liver. Gumma of the testicle.

**Six to Twelve Months** Iritis. Bossing of the skull on the frontal and parietal bones (Parrot's nodes or hot cross bun appearance) and cranio tabes (softened areas of bone in the skull) are probably rickety changes.

**Second Year** Dactylitis, the phalanges of the fingers or toes being swollen. Depression of the bridge of the nose (saddle bridge). Hydrocephalus and idiocy.

**Childhood and Later** Keratitis, especially between 6 and 12 years. The teeth. The central permanent upper incisors are notched at the cutting edge, which is narrower than the base, and the teeth are spaced (Hutchinson's teeth). The first permanent molars may be dome shaped, owing to failure of development of the central portion of the crown (Moon's teeth). Deafness may occur suddenly from gummatous destruction of the internal ear. Periostitis. The tibiae become curved forwards and thickened (sabre tibia). Painless swelling of joints, such as the knee (hydrarthrosis). Nervous lesions may be manifest, such as dementia, juvenile tabes or general paralysis. Diabetes insipidus may result from a basal syphilitic meningitis. The blood Wassermann reaction is usually positive up to puberty but may become spontaneously negative later in life.

The cerebro spinal fluid. This shows changes characteristic of syphilis (see p. 390) in about 40% of cases, depending upon whether the central nervous system is involved.

**Differential Diagnosis** Hutchinson's diagnostic triad is of value in childhood, the stigmata are interstitial keratitis, the typical incisor teeth and deafness. In infants a naphlin rash must not be mistaken for a syphilitic roseola. The history, appearances of the child and positive Wassermann reaction of the child and its mother are usually diagnostic.

**Course and Complications** The victim of congenital syphilis may

be very slightly or very severely affected. Fresh manifestations are liable to show themselves in late childhood or early adolescence, as mentioned above. Usually the child grows up, but is often stunted, and the intelligence may be below normal. Secondary infections are liable to occur.

**Prognosis.** This depends upon the intensity of the infection and the organs affected. The virulence usually diminishes in proportion to the length of time the mother has been infected.

**Treatment. Prophylactic.** The mother should be given a proper course of anti-syphilitic treatment before or during pregnancy.

**Curative.** The child should be treated directly the disease is recognised. Arsenic is injected intramuscularly into the gluteal region in the form of sulpharsphenamine. The following is the dosage: *Æt.* 1 to 3 months, 0.06 G., 6 weekly injections. *Æt.* 3 to 12 months, 0.12 G., 6 weekly injections, and *æt.* 1 to 3 years, 0.24 G., 6 weekly injections. 0.06 G. is dissolved in the ampoule in 1 mil. of freshly distilled water. Three such courses should be given with a 2 months' interval between each. Alternatively Stovarsol tablets (acetarsol B.P.Add.) may be given cautiously by mouth. For an infant weighing 9 lb. the dose is gr.  $\frac{1}{4}$  daily for the first week, gr.  $\frac{3}{4}$  daily for the second week, gr. 1 daily for the third week and gr.  $1\frac{1}{4}$  daily during the fourth to tenth weeks. The course is repeated three times, with 4 weeks' interval between each course. Simultaneously Quinostab 0.08 G. is injected intramuscularly weekly. Mercury is simultaneously administered, either by inunction as blue ointment (ung. hydrarg. B.P.), a piece about the size of a pea being rubbed daily for 15 minutes into different sites, such as the abdomen, the axillæ, the groins and the back, for 4 consecutive days each week. It may be given alternatively by mouth as pulv. hyd. c. creta gr.  $\frac{1}{4}$  nocte, with the addition of pulv. ipsecac. et opii gr.  $\frac{1}{12}$  if it causes diarrhœa.

### Acquired Syphilis

**Incubation Period.** This is usually about 28 days, probably never less than 10 days, but it may be prolonged to 12 or 13 weeks.

**Clinical Findings. Primary Stage.** The patient gives a history that about 4 weeks after sexual intercourse he noticed a small red spot on the penis. This enlarged, but was painless and gradually formed a sore.

**On Examination:** The sore after 2 to 3 weeks has an indurated cartilaginous feeling (hard or Hunterian chancre). Serum exudes from it on slight scarification, and this serum is teeming with treponemes. The chancre may be within the meatus of the urethra, or at other sites as mentioned on p. 560. If untreated the chancre heals in about 6 to 8 weeks, leaving a scar. The neighbouring lymphatic glands enlarge, and treponemes are found in the fluid obtained by gland puncture. The glands may suppurate if there is a mixed infection, constituting a bubo. In about another fortnight enlargement of glands is also noted at the back of the neck, under the arms and in the epitrochlear regions. The blood Wassermann reaction becomes positive about 8 weeks after

infection, and changes in the protein and cell contents of the cerebro spinal fluid are usually present

*The Secondary Stage* This is noted about 4 weeks or later (up to 6 months) after the appearance of the primary sore, and is characterised by systemic disturbance, as shown by headache, sore throat, anæmia, skin rashes, generalised glandular enlargement and at times by nephrosis. The patient may also complain of pains in the limbs due to synovitis or periostitis. Various rashes may occur, the syphilitic roseola is ham coloured or brownish, and is seen on the trunk and extremities. Brownish pigmentation with areas of leucoderma may develop on the neck, especially in dark haired patients. Papulo pustular or scaly eruptions may also appear on the body and limbs, the epithelium may become heaped up in a conical mass, forming rupia. Soft warty excrescences or condylomata are apt to form on moist surfaces which are not kept clean, as around the anal margin, vulva and under the breasts. The throat may be very red, and "snail track" greyish white streaks may be seen on the fauces or soft palate, and ulceration of the tonsils. White mucous patches may occur inside the cheeks. Alopecia of a general or patchy type is liable to occur. Chronic laryngitis may be present. Jaundice may develop with an enlarged liver, or in a severe form with acute yellow atrophy. The blood. There is anæmia. Treponemes may be present, and the Wassermann reaction is positive. The cerebro spinal fluid. The Wassermann reaction is often positive, and other changes such as excess of globulin, increase of lymphocytes and a characteristic colloidal gold curve (Lange's test) may be present (see Fig. 25).

*Tertiary Stage* This stage may emerge directly from the preceding one, or be separated from it by an interval of many years. It is characterised by the formation of gummata. The gummata may appear in various parts of the body. In the skin and subcutaneous tissues as ulcerating nodules which separate, leaving punched out ulcers, and on healing form thin paper like scars, in muscles forming painless swellings, which eventually ulcerate, and in bones, causing severe pains, usually intensified at night. Gummata may also form in internal organs such as the liver, the testis, lungs, pituitary and the adrenal. The heart may be involved, the gumma giving rise to disturbances of conduction according to its situation, or to localised myocardial degeneration. Syphilitic arterial changes give rise to cardio-vascular or renal degeneration. The various neurological diseases, such as general paralysis, tabes dorsalis, cerebral gumma, meningitis and meningo myelitis, are described in the chapter on nervous diseases. The alimentary tract may be affected. Glossitis, leukoplakia, localised gumma or ulceration may occur in the tongue. There may be ulceration of the tonsils or gumma formation in the œsophagus, less frequently in the stomach. Iritis and otitis may also occur as tertiary manifestations. A prolonged continued temperature lasting for several weeks may occur at times during the tertiary stage. The various results produced by tertiary syphilis are more fully considered in the chapters dealing with the heart, lungs, alimentary tract, etc.

**Differential Diagnosis.** In the primary stage the diagnosis should be established by finding treponemes in the serum from the chancre, and treatment should not be delayed until the blood Wassermann reaction becomes positive. Chancroid, herpetic and scabietic lesions may cause confusion. In the later stages the diagnosis is usually established by the Wassermann reaction of the blood and the cerebro-spinal fluid. Combined lesions may occur, such as an epithelioma of the tongue in a patient who is also a victim of syphilis. A positive Wassermann reaction may also be found during the pyrexial stage of malaria, although the patient is not suffering from syphilis, and it has been recorded in other diseases, such as scarlet fever, typhus fever, yaws, etc.

**Course and Complications.** The disease usually pursues a very chronic course, and the treponemes are apt to lie dormant in various parts of the body, shut off from the blood stream. Later they give rise to severe reactions in the form of tertiary lesions.

**Prognosis.** Syphilis is a very serious disease, and it is impossible to say in any individual case what course it will pursue. A mild initial lesion may be followed years later by crippling cardiac complications or fatal nervous affections. Efficient treatment in the earliest stages holds out the best hope of a permanent cure.

**Treatment. Prophylactic.** Avoidance of irregular sexual intercourse is the best preventive. Apart from this, protection is afforded by the use of a condom or sheath during intercourse and the immediate after-disinfection by the application of 83% calomel ointment in lanolin. Stovarsol tablets (acetarsol B.P.Add.) gr. 4, 4 every morning before breakfast, for 4 days, repeated the second and third week after exposure to infection, are also of value. The patient should not marry for 2 years from the beginning of the treatment, and his Wassermann reaction should be negative.

**Curative.** As soon as the chancre appears and the diagnosis is established by finding treponemes in the smear, a complete course of treatment should be instituted. The patient must not take any alcohol, and if feverish he should be in bed. Treatment consists in intravenous injections of an arsenic preparation, and intramuscular injections of a bismuth derivative.

The arsenic preparation neoarsphenamine contains about 20% arsenic, and is a yellow powder. It is put up in ampoules containing the required dose. It must be protected from air and injected immediately after being dissolved, the solution not being exposed to air. 2 to 5 mils of freshly distilled water are used to dissolve the powder in the ampoule, by drawing it in and out of the syringe; it is then immediately injected slowly into a vein in the arm. The usual initial dose for an adult male is 0.3 G., the subsequent doses being maintained at 0.6 G.

A bismuth preparation such as Quinostab (0.1 G. per mil.) is injected intramuscularly into the upper and outer quadrant of the buttock, using a needle, 22 S.W.G., 1½ to 2½ inches long. Care must be taken that it is not injected into a vessel, or pulmonary embolus may occur, 6 or 8 hours later.

*First Course* This lasts 10 weeks and is as follows. Weekly injections of neoarsphenamine beginning with 0.3 G. and followed by 0.6 G., 10 injections in all, being a total of 5.7 G. NAB or 1.14 G. arsenic. The Wassermann reaction is then determined and no treatment is given for 4 weeks. Twelve weekly injections of 0.3 G. of Quinostab are now given, the total amount of bismuth thus injected being 3.6 G. The Wassermann reaction is now taken again and the patient is given another 4 weeks without treatment.

*Second Course* This begins at the end of the first 30 weeks. Five weekly injections of 0.6 G. of neoarsphenamine are given, with a total amount of arsenic of 0.6 G. The Wassermann reaction is then determined again and there is a further interval of 4 weeks without treatment. A second course of 12 weekly injections of Quinostab is now given, total bismuth in the course = 3.6 G. After another 4 weeks' interval without any treatment, 12 more weekly injections of 0.3 G. Quinostab are given (total bismuth = 3.6 G.). The Wassermann reaction is now tested again.

*Third Course* After another 4 weeks' interval 5 weekly injections of 0.6 G. of neoarsphenamine are given (total = 0.6 G. arsenic). After a further 4 weeks' interval without treatment a final course of 12 weekly injections of 0.3 G. of Quinostab is given (total = 3.6 G. bismuth).

The whole course of treatment therefore lasts for 92 weeks, the amount of arsenic injected intravenously is 2.34 G. and the total amount of bismuth is 14.4 G. The actual number of injections required must vary with the age and sex of the patient and with the stage of the disease at which treatment is begun, thus less treatment is usually required in an early primary case in which the blood Wassermann is negative than in a later case in which it is positive. In many cases, in which the disease has been present for over 2 years, it is impossible to obtain a negative Wassermann reaction by treatment. In such cases artificial production of pyrexia by intravenous injection of typhoid mixed vaccine (TAB) is for the non specific treatment of arthritis (see p. 612), followed by a further course of neoarsphenamine, will sometimes convert the Wassermann reaction to the negative. In a satisfactory case the blood and cerebrospinal fluid Wassermann reactions should be negative at the end of a year, both before and a week after giving a provocative injection of 0.45 G. of neoarsphenamine. This test should be repeated in 6 months, and a year, and if the reaction is positive another course of neoarsphenamine and bismuth should be given.

Continuous treatment is preferred by some authorities, no rest periods being given. The bismuth injections follow immediately after the neoarsphenamine course, which is again directly followed by more bismuth injections. The whole course thus lasts 74 weeks, with four series of neoarsphenamine and four series of bismuth injections. This method is especially valuable if given during the first year of the disease.

*The Use of Mercury in Treatment* Intramuscular injections of mercury have now been largely superseded by bismuth, as the latter appears to have greater spirochæticidal properties.



**Precautions during Treatment.** The injection of neoarsphenamine should be given fasting. 1 oz. of dextrose in a glass of lemonade should be given 1 hour before the injection. The bowels should be kept opened daily. The skin, especially in the flexure of the elbows, must be inspected before each injection, to see if there is any sign of dermatitis. If so, the neoarsphenamine must not be repeated and injections of sod. thiosulphate should be given, such as Thiostab, 0.45 G. in a sterile 10% aqueous solution intravenously daily for 6 doses. Toxic effects of bismuth are shown by the appearance of stomatitis, a purple or blue line on the gums near the teeth, and by albuminuria. The urine should be tested before each injection for albumin.

**Contra-indications.** Neoarsphenamine should be given with caution if there is albuminuria, but it is not absolutely contra-indicated, as the albuminuria may be due to a syphilitic nephrosis. If there is glycosuria, small doses of neoarsphenamine should be used. Specific insulin treatment will also be required if there is hyperglycemia (see Diabetes mellitus, p. 631). Neoarsphenamine is contra-indicated in syphilitic diseases of the liver, and in cardio-vascular degenerations it should only be used in small doses, such as 0.3 G. It is contra-indicated also in hæmophilia and in Addison's disease.

**Reactions.** Various reactions may occur during treatment by injection of neoarsphenamine:

**Immediate.** Anaphylactoid or nitritoid reactions. During or directly after the injection the patient may complain of dyspnœa, faintness, pain in the gums or teeth and a taste of garlic; the face flushes, the pupils dilate and unconsciousness may supervene. One ml. of liq. adrenalin. hydrochlor. solution should be injected intramuscularly.

**After a Few Hours.** A toxic reaction may occur with headache, shivering or a rigor, rise of temperature, nausea, vomiting and diarrhœa. There may be pain in the back and cramps in the legs. An urticarial rash may appear. Prophylactic treatment consists in giving dextrose before the injection.

**The Jarisch-Herxheimer Reaction.** This may occur on the day after the first or second injection. It is probably due to an increased activity around the site of the lesions, caused by liberation of toxins from the treponemes destroyed. Thus the rash may be more marked and the Wassermann reaction become more strongly positive. If the lesions are cardiac, laryngeal or cerebral, very grave results may ensue from vascular changes in the affected areas, such as heart failure, laryngeal obstruction, or paralysis.

**Later Reactions.** These include: Cutaneous, exfoliative dermatitis. Alimentary, stomatitis and diarrhœa. Hepatic, jaundice and atrophy of the liver. Renal, albuminuria and nephrosis. Nervous, headache, convulsions, or mental changes. Pancreatic, diabetes mellitus. General, insomnia and loss of weight. Agranulocytosis or aplastic anæmia at times occur.

For the cutaneous reaction calamine lotion should be applied locally, and intravenous injections of sodium thiosulphate given, 0.45 G.

daily for 3 or 4 doses or intramuscular injections of Contramine 0.125 G on alternate days up to 6 doses

Venesection and lumbar puncture may relieve the nervous symptoms. For severe jaundice dextrose oz. 4 and alkalis such as sod. bicarb. gr. 20 t.d.s. should be given by mouth daily, and 0.45 G sodium thiosulphate intravenously daily for 10 doses.

**Bismuth Reactions** In all cases undergoing treatment with this metal a watch should be kept for overdosage, as shown by stomatitis, a blue line on the gums, albuminuria or colitis. If these appear the treatment must be stopped and injections of Thiostab given (see p. 572). In any case the teeth should be put in good order before the injections are given.

### Gonorrhœa

**Definition.** A venereal disease caused by infection with a specific micrococcus.

**Etiology** The cause is the *Neisseria gonorrhœa* (gonococcus) of which there are many strains. Infection in man is usually by sexual intercourse. Infants may have their eyes infected at birth if the mother is suffering from the disease. Gonorrhœal vaginitis of children may become epidemic in institutions due to infected towels or clothes.

**Pathology** The gonococcus penetrates mucous membranes, such as line the urethra or vagina. It may spread directly to the uterus, Fallopian tubes, peritoneum, glands of Bartholin, prostate, seminal vesicles, bladder, pelvis of the kidney or rectum. It may be carried by the blood stream to the heart, meninges, joints, muscles, tendons or eyes. The toxins may cause such lesions as peripheral neuritis, keratoderma or iritis. Infection may be conveyed by the finger to the eyes, causing ophthalmia.

**Incubation Period.** This is usually from 3 to 10 days.

**Clinical Findings** Acute gonorrhœa is a disease which is usually classified as "surgical," and will not be dealt with here. From the medical aspect gonorrhœa is of importance as causing acute arthritis, meningitis or myelitis, pericarditis, endocarditis and myocarditis, peripheral neuritis and septicæmia.

**Gonorrhœal Arthritis** The patient is usually a young adult of either sex who may be suffering from an attack of acute gonorrhœa, or who may have been infected previously. He is suddenly taken ill with malaise and pain in one or more joints. Often one joint only is affected, such as the knee, ankle, wrist, shoulder or elbow, or the pain may be localised to the sterno-clavicular, temporo-mandibular, spinal or sacro-iliac joints.

**On Examination** The affected joint, if one of the large ones, is swollen, tender, and the skin over it may be slightly red and hot. Effusion is present in the joint and movement is painful. There is also constitutional disturbance, for the tongue is furred and the temperature and pulse rate are raised. If fluid is aspirated from the joint it is sero-fibrinous and may contain gonococci. In women there is usually a

vaginal discharge, and in men a urethral discharge may be present before or after prostatic massage. A swab taken from the cervix uteri or from the urethra after prostatic massage often shows gonococci.

*Varieties.* Chronic hydrarthrosis, which is painless and insidious in onset, may occur, or subacute polyarthritis.

*Differential Diagnosis.* The commonest error is to diagnose the condition as acute rheumatism, or some other variety of acute infective arthritis. The characteristic features are the history of gonorrhœa; which, however, is often not forthcoming, the special joints affected, the presence of gonococci in the genito-urinary discharge, a positive complement fixation test, and the response to specific treatment. Salicylates fail to give relief.

*Course and Complications.* The joints usually recover with proper treatment, but stiffness may persist for long periods. A chronic, painless arthritis, with effusion, may ensue in some cases.

*Prognosis.* This is generally good.

*Treatment.* It is hoped that adequate and early treatment of gonorrhœa with Sulphapyridine (M & B 693) or Sulphathiazole (M & B 760), together with daily urethral irrigation with 1 in 8,000 potassium permanganate solution or vaginal douches of 1 in 4,000 solution, will result in cure, and prevent the medical complications and sequelæ enumerated above. In gonorrhœal arthritis Sulphapyridine or Sulphathiazole may be given in doses of 1 G. every 6 hours for a week. Massage and passive movements should be applied to the joints as soon as the pain subsides. The other medical gonorrhœal affections are described under their respective headings.

### Septicæmia

*Definition.* An illness due to the circulation and multiplication of micro-organisms in the blood.

*Etiology.* Septicæmia is usually due to the streptococcus hæmolyticus, the streptococcus viridans in subacute bacterial endocarditis, or to staphylococci or pneumococci. Other organisms include the enterica and dysentery group bacilli, the Neisseria gonorrhœa (gonococcus), the Neisseria meningitidis (meningococcus), and the Bacillus anthracis.

In bacteriæmia the organisms are present in the blood, but do not give rise to symptoms. The organisms may gain access to the blood from many sites, such as the nose and throat, the roots of teeth, the alimentary tract, the skin, the uterus, a post-mortem wound, a boil, or a bone in osteomyelitis.

*Pathology.* The normal phagocytic function of the leucocytes appears deficient. Post-mortem, the spleen is enlarged and soft, the intima of the aorta is usually stained pink, and petechiæ may be seen on the pleuræ and pericardium.

*Clinical Findings.* There may be a history of a wound at an operation or post-mortem examination, of a boil, a carbuncle or erysipelas, or of an operation, such as curettage of an infected uterus. Usually in about

36 hours the patient suddenly feels ill with headache, shivering and perhaps a rigor. Chronic meningococcal septicæmia is described on p 293

**On Examination** The pulse is rapid, 100 to 120, and may be irregular owing to premature systoles. The temperature is high and intermittent or remittent in type, reaching 103° to 105° F in the evenings and falling to 97° to 99° F in the mornings. The tongue becomes dry and brown, petechial spots may be seen in the skin, or zones of bright erythema or a scarlatiniform rash. Pain and swelling may occur around various joints. The spleen may be just palpable. The patient becomes more drowsy and often delirious. Pleural or pericardial friction may be heard. The abdomen often is distended owing to flutulence, and the urine contains albumin and perhaps blood due to a focal nephritis. The blood. The polymorphonuclear cells are usually increased. The blood culture is generally positive.

**Differential Diagnosis** This is made by the history, clinical findings and a positive blood culture. In pyæmia, local abscesses form in various parts of the body. Other conditions giving rise to fever, such as influenza, enteric fever, tuberculosis and acute rheumatism, may have to be excluded.

**Course and Complications** In virulent infections death usually occurs in a day or so, low grade infections cause prolonged fever. Pericarditis, pleurisy and focal nephritis may be considered as complications.

**Prognosis** Septicæmia is always a serious condition but the chance of recovery has been doubled by the use of the sulphonamide drugs in adequate dosage.

**Treatment.** The patient must be in bed and well nursed. He should be encouraged to drink plenty of bland fluids, such as water, barley water and lemonade. He should have fresh air and the skin should be sponged several times daily. The bowels should be opened daily with salines (mag. sulph. gr 60 to 120 in water, or Mist Alba (B.P.C.) fl oz 1 mane). Sleep should be secured with paraldehyde (m 15 capsules), 4 or 8 as required. Any local septic focus should be treated with fomentations, and if pus forms it should be let out where it points through a small incision, care being taken not to spread the infection into the blood stream by an extensive or deep operation. If the infection is due to hæmolytic streptococci sulphanilamide should be given, 4 G repeated in four hours and followed by 1 G every 4 hours for 4 or 5 days. Sulphapyridine (M & B 693) should be used for streptococcus viridans infections in similar doses. Sulphathiazole (M & B 760) should be given for staphylococcal infections the initial dose being 4 G, followed by 2 G every 4 hours until a total of 30 G has been given. Chronic meningococcal septicæmia is best treated with Sulphapyridine in doses of 4 G followed by 2 G 4 hours later, and then 1 G t.i.d. for 3 to 4 days. Stimulants in the form of Coramine (nikethamidum B.P. Add.) 15 ml or strychnin hydrochlor gr 1/60, may also be required as hypodermic injections, or liq. adrenal hydrochlor m 5 every 4 hours.

## Pyæmia

**Definition.** A condition characterised by the circulation and multiplication of micro-organisms in the blood, and the formation of metastatic abscesses.

**Etiology.** The organisms are usually the staphylococci, streptococci or the *Bacterium commune* (*B. coli*). They gain access to the circulation from some focus such as an infected wound, a boil or carbuncle, osteomyelitis, bronchiectasis, calculous pyelitis, appendix abscess, an abscess in the rectum or gall-bladder, or a perinephric abscess. Lack of asepsis may be the cause in a patient who is giving himself insulin injections.

**Pathology.** Abscesses may form under the skin, in muscles, in joints, and in internal organs, such as the brain, lungs, or liver, due to septic infarcts. Infarcts may also occur in the spleen; purulent effusions may form in the pericardium or pleural sacs. Multiple small abscesses may develop in the kidneys under the capsule.

**Clinical Findings.** Three main types are described: Systemic venous; Arterial; Portal.

**Systemic venous pyæmia.** An illustrative case is as follows: A young man, apparently in good health, may have had a series of boils which have healed. He is suddenly taken ill with pain in the knee and thigh, which increases in severity and prevents movement.

**On Examination:** The knee is a little swollen and very painful on movement. The temperature is raised to about 100° F. and the pulse is about 100. The patient becomes more ill, has headache, shivering attacks, and the tongue is white with a thick fur. An urticarial rash appears upon the body. The temperature rises higher to 101° or 102° F. and the pulse to 120 or 130. Tenderness is now felt in the thigh and just above Poupart's ligament (the inguinal ligament). Pleural friction is heard at the base of one lung. The blood: There is a leucocytosis of 17,000 per c.mm., with 90% polymorphonuclear cells. Culture yields a pure growth of staphylococcus aureus. The urine: This is acid, containing a trace of albumin, some white and red blood cells and granular casts. Staphylococcus aureus is present in a catheter specimen. The whole leg now becomes swollen and a fluctuating swelling is felt in the groin, above and below Poupart's ligament. The patient becomes cold and clammy, although he says he feels very hot, and he dies in about 8 days from the onset, with pyarthrosis of the hip joint.

**Differential Diagnosis.** This is as for septicæmia (see p. 575).

**Course and Complications.** The course in fatal cases is usually rapid, death occurring in a week or so. Recovery may occur after many weeks in some instances. Complications include the formation of septic infarcts and abscesses in various parts of the body.

**Prognosis.** This is often hopeless, but recovery may occur.

**Treatment.** This is as for septicæmia (see p. 575). With local abscess formation the results are usually disappointing. Concentrated staphylococcus antitoxin-globulins may also be tried. A preliminary desensitising dose of 0.1 mil. of the serum, diluted 1 in 10 in warm saline, is injected intravenously, followed half an hour later by the intravenous injection

of 40 mls of warmed undiluted serum. Local abscesses should be opened. Blood transfusion is beneficial if there is anæmia.

*Arterial pyæmia*. This is exemplified by malignant endocarditis, with metastatic abscess formation (see p. 236).

*Portal pyæmia*. The infection is conveyed by the portal veins to the liver, with abscess formation (see p. 75).

### Rheumatic Fever

#### (*Acute Rheumatism*)

**Definition.** An acute disease characterised by fever, joint pains and a liability to carditis.

**Etiology.** The view gains ground that rheumatic fever is due to infection through the throat and possibly through the intestine with a streptococcus, usually hæmolytic, the organisms passing by the blood stream to the sites of election, the heart, the joints, subcutaneous tissues and meninges. Some authorities believe that streptococci lodged in sites such as the tonsils, produce a chronic allergic state, which on stimulation results in acute rheumatism. Spread in some cases may be due to droplet infection. An alternative view is that the virus is ultramicroscopic. *Predisposing causes*. 1 Familial diathesis. The disease runs in families, presumably due to deficient resistance, and the case incidence points to contact infection. 2 Age and sex. Children and young adults are chiefly affected, females preponderating somewhat in childhood and males later. 3 Social factors. Overcrowding, poor food (possibly lacking in vitamin C) and damp houses are of importance. The majority of cases are in the hospital class of patient. 4 Climate and season. Rheumatic fever prevails chiefly in temperate climates, in England especially in the autumn and spring, and epidemic years occur. Statistics tend to show that the incidence of rheumatism in children corresponds closely with that of rainfall. 5 Previous attacks. These predispose to subsequent ones, an epidemic of tonsillitis may precede one of acute rheumatism.

**Pathology.** It is not definitely known if the causative organisms circulate in the blood and settle in the joints and heart, or whether the lesions are due to a toxæmia, the organisms present from time to time in the blood and tissues being secondary infective agents. *Post mortem*. The heart. Pericarditis, dry or with effusion, may be present. Aschaff's sub miliary nodules may be found microscopically in the myocardium. They are spindle shaped and contain connective tissue cells and fibroblasts. Small sessile vegetations may be seen on the valves, such as the mitral or aortic valve. Subcutaneous nodules felt before death may not be found at autopsy, these are due to an inflammatory exudate, but in some cases they are fibrosed and then persist after death. Streptococci have been grown from the pericardium and from the affected heart valves and from subcutaneous nodules.

**Clinical Findings.** The patient is usually a child or young adult. In children the onset is often quite insidious. Many children suffer from subacute rheumatism, the chief symptom of which is so called

growing pains, which come and go and are felt in the limbs or back. They may also complain of attacks of abdominal pains. If the patient is an adult, he may have noticed an attack of acute pharyngitis 10 to 28 days previously, with malaise and fleeting limb pains. He is then suddenly seized with acute pain in one or more joints or in the præcordium. A history of similar previous attacks may be given.

*On Examination:* The patient is usually pale and not infrequently has auburn hair. Sweating is a characteristic feature; a sudaminal rash may be seen and the sweat has a rather sour smell. The tonsils are often unhealthy; enlarged, pitted or fibrosed. Evidence of infection may be seen around the teeth, and a dental radiogram may show apical infection. Usually the larger joints are affected, such as the ankles, knees, elbows and shoulders. They may appear normal or be definitely swollen. This swelling is due either to periarticular œdema or to fluid in the joints. Fluid removed from a distended joint is usually sterile. The skin over the joint is either normal in colour or a little red and hot, and there is tenderness on palpation, any attempt to move the joint causing very severe pain. The changes may spread rapidly from joint to joint, one improving as another is involved. In children only one joint may be affected. This is painful and usually swollen. The temperature is usually over 100° F., and rises as fresh joints are affected. Afebrile periods may occur, during which the pulse remains frequent. The heart: Rheumatic carditis is present in almost every severe case of rheumatic fever, although clinical evidence of this may be lacking in some instances. If there is no cardiac enlargement and no murmur is heard there is no clinical evidence of cardiac involvement. The rate of the heart, the position of the apex and the presence or absence of murmurs should be recorded daily, together with the character of the heart sounds. Special attention is paid to the tone of the first sound at the apex and whether or not the second sound is reduplicated there. The apex beat may move out slightly and a localised systolic murmur be heard there owing to dilatation. As the condition improves the apex beat may return to its normal position and the murmur disappear without there being any indication of permanent cardiac damage. In other cases the systolic murmur may persist, later a diastolic murmur appears, indicating valvular damage. Indications that the heart is definitely affected are the presence of a mid-diastolic, an early diastolic, or a presystolic murmur, localised usually to a small area internal to the apex beat. It is very difficult to hear the early or mid-diastolic murmur unless the heart rate is slow. If the heart recovers, the early and mid-diastolic murmurs may disappear and also the systolic murmur. These early and mid-diastolic murmurs are probably produced by the ventricle sucking the blood through an inflamed mitral valve. An aortic diastolic murmur is less commonly heard. In some cases there may be a dry pericarditis which occurs early in the disease and is associated with pain, restlessness, vomiting and fever. An electrocardiogram may show prolongation of the P-R interval, indicating a degree of heart block, which is restored to normal as the patient recovers.

Rheumatic nodules are felt chiefly in children, indicating that the

disease is still active, and they are often associated with carditis. They are about  $\frac{1}{4}$  inch long, and are found under the skin, especially over the knees, elbows, occiput, scalp, backs of the hands and back of the chest. The tissues should be put on the stretch to feel them. They often come and go quite rapidly.

The urine is diminished, high coloured, and may contain a trace of albumen. The blood culture is usually sterile, but diplococci have been found in some cases, there is a leucocytosis in the acute stages and anæmia may rapidly appear. The rate of sedimentation of the red cells is increased during the active stages of the disease.

**Differential Diagnosis** Acute rheumatism must be distinguished from other forms of acute infective arthritis such as acute osteoarthritis and acute rheumatoid arthritis, and from gout, the response to salicylates in acute rheumatism is a good therapeutic test.

In children acute rheumatism may be diagnosed when in reality the illness is due to osteomyelitis, in the latter the pain and tenderness are near to, rather than in, a joint and they are localised to one spot. Scurvy and poliomyelitis, owing to the pain or immobility of the limb, may also lead to confusion, if the patient is not examined thoroughly.

**Course and Complications** The course is usually rapidly modified by the administration of salicylates, the temperature falling to normal in a week or 10 days. There is a tendency for the pains to pass from joint to joint and for symmetrical joints to be affected. Relapse may also occur with a rise of temperature and recurrence of pain in the joints previously affected. The heart is the danger spot in acute rheumatism, the apex beat may pass outwards a little owing to dilatation, a soft apical systolic murmur being heard, due to dilatation of the mitral valve. This probably indicates some myocarditis. Rheumatic carditis may be definitely diagnosed if there is cardiac enlargement, a systolic murmur at or near to the apex, an accentuated pulmonary second sound and an accentuated first sound at the apex. An aortic diastolic murmur also indicates carditis. Pericarditis or pericardial effusion may develop. Other complications include chorea, and hyperpyrexia (cerebral rheumatism), the temperature rising to over  $107^{\circ}\text{F}$ . Pleurisy is a rare complication. The important sequelæ are cardiac lesions, such as chronic endocarditis, usually of the mitral valve, leading to stenosis, or affections of the aortic valve such as regurgitation. Pericardial adhesions may result from pericarditis. Subsequent attacks of rheumatic fever are not uncommon.

**Prognosis** The disease is rarely immediately fatal, and the type is becoming progressively less virulent in England. Chorea is an unfavourable complication. The ultimate prognosis depends upon the presence and severity of the cardiac lesions.

**Treatment** *Prophylactic* Unhealthy tonsils should be removed and the teeth well cared for, damp clothes should be changed as soon as possible. The presence of slums and deficiency in diet are factors which call for correction.

*Curative* The patient should be put to bed between blankets or woollen sheets, and kept lying down with only one pillow. He must



remain thus in bed until the temperature has been normal for 3 weeks after the salicylates have been discontinued, providing there is no cardiac affection. The sleeping pulse rate and the sedimentation rate of the red cells must also be normal. After this additional pillows may be given, and the patient gradually got up, providing the pulse rate is not accelerated or the temperature raised. If the heart is involved he should be kept in bed for a minimum of 3 months (see Pericarditis, p. 205). Sod. salicyl. gr. 15, sod. bicarb. gr. 30, syr. aurant. m. 30, aq. ad fl. oz.  $\frac{1}{2}$  should be given every 3 hours for 4 doses and then every 4 hours. When the effect of the drug is produced, as shown by relief of pains and fall in temperature, the dosage is reduced to six-hour intervals and then to 3 times a day. If toxic symptoms occur, such as marked buzzing in the ears, nausea, vomiting or hæmaturia, or if the pulse falls below 60, it should be further reduced or discontinued. If sodium salicylate cannot be tolerated, the pure natural preparation should be tried. If this disagrees salicin gr. 10 to 20 may be given t.d.s.

The affected joints should be covered with gaultherium ointment (ung. methyl. salicyl. B.P.C.), wrapped in wool and protected from the pressure of the bed clothes by a cradle. The bowels should be opened by calomel gr. 3, and mag. sulph. gr. 120 next morning, and subsequently regulated as necessary with aperients. If there is much pain at the onset pulv. ipecac. et opii gr. 10 should be given at night to secure sleep. The diet should be fluid during the febrile stages, such as imperial drink, barley water, dextrose orangeade, and milk, but not meat extracts. The diet is later increased with bread and butter and semi-solids. It is advisable not to give meat until convalescence is fully established. Subsequently the tonsils should be removed if they are septic, causing enlargement of glands in the neck and repeated sore throats. Removal of the tonsils will not, however, prevent the occurrence or re-occurrence of rheumatism in children. Dental sepsis should also be eradicated. The treatment of pericarditis is described on p. 205. Owing to the prolonged rest required children are best sent, after the acute stage, to special recovery homes and later to open-air or residential schools. These should not be by the seaside or near a river. Tropical or sub-tropical climates are most suitable. Ferrated emulsion of cod-liver oil m. 30 to 60 t.d.s. should be given to children during convalescence.

### Influenza (La Grippe)

**Definition.** An acute disease of doubtful etiology, characterised by fever, prostration, a great liability to pulmonary complications and to epidemic incidence.

**Etiology.** The *Hæmophilus influenzae* (Pfeiffer's bacillus) is not usually accepted as the cause of influenza, and there is no definite proof that it is due to a filter-passing virus pathogenic for ferrets and recoverable from the garglings of patients suffering from influenza. Streptococci are probably secondarily infecting organisms. *Predisposing causes:* Nothing is known on this score except that the disease tends to occur

in pandemics about every 10 to 40 years, and in epidemics about every 33 weeks. In the 1889 pandemic infants and old people were affected, whereas in the 1918 pestilence young adults in the prime of life were the victims. Three waves were noted in the last two pandemics, the second one being the most serious. The disease spreads with great rapidity.

**Pathology.** Post mortem the changes resemble those of a hæmorrhagic septicæmia, but the spleen is not enlarged. Hæmorrhages may be seen in the sheath of the rectus abdominalis and in the muscle itself. The pericardium may contain a little blood stained fluid. The trachea shows a typical pink colour of its lining membrane between the rings, especially in the lower part. The bronchi contain mucopus. Exudation may be seen in the bronchioles and alveoli, which prevent proper aeration of the blood. Hæmorrhagic areas of lung tissue, which may float or sink in water, are typical, with patches of collapsed lung. Petechæ may be seen under the pleuræ, yellow or blood stained fluid may be found in the pleural sacs. The bronchial glands are enlarged and may show hæmorrhages. Petechial hæmorrhages are frequent in the mucous membrane of the stomach, and the interior of the ileum, cæcum or pelvic colon may be intensely congested. The kidneys are usually a little enlarged and congested.

**Incubation Period.** This is probably about 48 hours.

**Clinical Findings.** *The Mild Type.* The patient can often say the hour at which he was suddenly taken ill, with lassitude or severe prostration and at times shivering. Pain may be felt in the head, behind the eyes and on moving the eyes, under the sternum, along the diaphragm attachment, in the back, in the calves or shins and occasionally in other parts of the body. The patient may feel sick, have epistaxis or vomit.

**On Examination.** The conjunctivæ are often injected ( 'pink eye' ), the face flushed and the skin dry. The temperature rises rapidly to about 103° F and the pulse is moderately rapid. There is usually cough on the second day and the voice may be hoarse, even in mild cases. Careful examination of the lungs usually reveals slight abnormalities such as weak breath sounds at a base, or a few basal rales or some scattered rhonchi. A scarlatiniform rash may occur on the arms, body and legs, but the flexures are usually left clear. The blood shows a leucopenia. The urine usually contains no albumin. The temperature falls to normal about the third or fifth day, according to the type of epidemic prevailing and convalescence is rapidly reached.

**Severe Cases.** *The Bronchopneumonic Type.* The onset is similar to that of a mild case and it is impossible to say in any instance whether the patient will rapidly become dangerously ill. The severe symptoms may be due to an acute toxæmia, the temperature rapidly rising to 104° F or higher and the patient becoming cyanosed and dying in the course of a few hours. In other cases bronchopneumonia or a severe purulent bronchitis may develop in a few days. The respirations become rapid and may reach 50 to 60 a minute, but the pulse is not usually proportionately frequent, 110 to 120 being an average rate, and the tem

perature varies from 100° to 103° F. or higher. A peculiar heliotrope cyanosis is typical, in which the face and ears are mauve, but they may be leaden in colour or the face may be pale, with lips and ears blue. The respirations though rapid are not difficult and there is no orthopnoea. The cough is often troublesome and the sputum is of varying types, being either white and frothy, green and purulent, or tenacious and containing bright red or brown blood. Examination of the lungs often shows very slight signs, but generalised rhonchi may be heard, or there may be small areas of dulness, with weak breath sounds and crackling râles. The blood is "sticky," the red cells may number 8,000,000 per c.mm., and it is difficult to bleed a patient. There is usually a leucopenia of about 3,000 to 4,000 white cells per c.mm., with about 50 to 60% of polymorphonuclear cells. The urine may contain a trace of albumin; a few red and white blood cells are often found and occasionally a hyalo-granular cast. Fatal cases often become delirious towards the end, with low muttering. Patients who recover may complain of attacks of giddiness during convalescence.

*The Gastro-intestinal Type.* Other cases assume a gastro-intestinal form, with vomiting and diarrhoea, and bright blood may be passed in the motions.

*Differential Diagnosis.* A mild case of influenza is differentiated from an ordinary feverish cold by the greater prostration and muscular pains in the back, limbs and external ocular muscles, and also by its occurrence during an epidemic. Abroad it must be distinguished from such diseases as malaria (see p. 677) or dengue (see p. 704). The pulmonary varieties would not usually be diagnosed unless occurring in an epidemic, but the heliotrope cyanosis and tendency to hæmorrhages are suggestive.

*Course and Complications.* The course is very variable, as described above. Complications include: Bronchopneumonia, sterile pleural effusions, empyema, subcutaneous emphysema, herpes facialis, meningitis, sinusitis, otitis media, mastoiditis, jaundice, neuralgia and acute thyroiditis. A latent tuberculous focus in the lungs may be activated, and pulmonary fibrosis or bronchiectasis may ensue as a sequela. Profuse generalised sweating sometimes follows an attack of influenza and persists for a week or so.

*Prognosis.* Mild cases recover. Between 20 to 40% of severe cases develop bronchopneumonia; and of these about 40% prove fatal. Cyanosis is a very grave omen, and cases seldom recover if the respiration rate exceeds 50. Profuse sweating is a very favourable sign in severe cases.

*Treatment. Prophylactic.* During an epidemic all crowded places should be avoided. The throat should be gargled night and morning with 1 in 4,000 solution of potassium permanganate. Vaccination with a mixture of Pfeiffer's bacillus, pneumococci and streptococci or with epidemic influenza virus suspension is not of proved value. Attendants on patients should wear a gauze mask.

*Curative.* The patient should be isolated in bed in an airy room at the first symptom, and remain there until the temperature has been normal

for 2 or 3 days in a mild attack. Returning to work too early involves a risk of a serious and perhaps fatal relapse. The bowels should be opened with calomel gr 3 at night and mag sulph gr 120 next morning. For the muscular pains salicin gr 10 can be given in a cachet t d s or aspirin gr 5 t d s. The diet should be fluid during the fever stage, as for lobar pneumonia (see p 142) with plenty of bland drinks, over 5 pints a day should be taken to eliminate toxins. Linct codein (B P C), m 30 to 60, may be given t i d to relieve a dry and exhausting cough. If there is severe cyanosis oxygen should be administered continuously through a nasal catheter or B L B mask (see p 143). In influenzal pneumonia, if there is a leucopenia, leucocytosis may be stimulated by the daily intramuscular injection of 0.7 G pentnucleotide in 10 mls ampoules, the response being checked by a daily white cell count. Cardiac or respiratory stimulants, such as Coramine (nikethamidum B P Add) 1.5 ml, digitalin gr 1/100 or strychnin hydrochlor gr 1/60, may be given by subcutaneous injection, if required. There is no specific remedy, in a controlled series of cases massive doses of salicin repeated intravenous injections of 0.01 G of perchloride of mercury in 1 ml of water, and the prophylactic mixed vaccine, have been tried without any beneficial results, the best effects were obtained with the perchloride of mercury. The diarrhoea is treated by giving a powder containing pulv ipecac et opii gr 5 and bismuth salicyl gr 10 three times a day.

During convalescence a tonic such as Liq strychnin m 2, syr ferri phosphat co m 60 may be given t d s, and no strenuous exercise should be taken which might cause the heart to dilate.

### Tetanus

#### (Lockjaw)

**Definition** An acute disease, due to a specific bacillus, characterised by violent and painful muscular spasms.

**Etiology** Tetanus results from infection with the *Clostridium tetani* (B tetani), an anaerobic organism, which forms very resistant spores. The bacilli occur in the intestines of animals, such as the horse and cow. Man is infected through a wound being contaminated with soil containing the spores. The soil of Flanders was heavily infected during the 1914-18 war. Catgut used at an operation or wool used for dressings may contain the spores.

**Pathology** The bacilli remain localised, but give rise to very potent exo toxins. These are probably absorbed from the end plates of motor nerves in the muscles and pass along the nerves to the central nervous system, where they increase the excitability of the synapses. *Post mortem*, muscles, such as the rectus abdominalis, may be found ruptured. There are very slight changes seen in the central nervous system.

**Incubation Period** This is usually about 12 days. In acute cases it may be as short as 2 days or prolonged to several months, especially if a prophylactic injection of antitetanic serum has been given.

**Clinical Findings** The patient may give a history of a wound

when gardening, or there may have been a war wound or a compound fracture, or he may be convalescent from an operation. Tetanus is now more often seen in association with trivial than with severe wounds, owing to the generalised use of antitoxin in the latter. In some cases tetanus appears to arise idiopathically, but the organism then presumably enters the body through an abrasion. In tetanus neonatorum infection occurs through the severed umbilical cord. The patient first notices muscular stiffness, affecting the jaw, neck or extremities. In the course of a few hours the stiffness increases and painful cramps may occur in the affected muscle groups, with dysphagia. In children convulsions may be the first manifestation of the disease.

**On Examination:** In the early stages no abnormality may be found except an increase of the deep reflexes, such as the knee-jerks. In a developed case the picture is most striking and terrible. The patient has frequently recurring seizures of violent muscular contractions, so that the body may be arched backwards, resting on the occiput and heels (*opisthotonus*), or bent forwards (*emprosthotonus*), or twisted sideways (*pleurothotonus*). The jaw muscles may be firmly contracted (*trismus*) and contraction of the facial muscles produce the appalling "risus sardonicus." Trismus is rarely combined with facial paralysis. Any slight external stimulus such as a noise, a bright light or a touch may provoke an attack. The mind remains clear and the patient may cry in agony or be unable to articulate owing to spasm of the intercostal and respiratory muscles. The skin is moist or sweating, but the temperature and pulse are usually normal. The temperature may rise to 108° F. in the terminal stages, although the patient is free from spasms. The blood: A leucocytosis of about 12,000 per c.mm. may be present. The cerebro-spinal fluid is usually normal, but under increased pressure. In local tetanus the spasms are limited to a group of muscles. This occurs especially some months after a bony injury. In cephalic tetanus there is dysphagia and paralysis of cranial nerves, together with generalised convulsions.

**Differential Diagnosis.** A clue to the diagnosis is usually afforded by the history. Other causes of trismus (see p. 369) must be excluded, and spasms due to strychnine poisoning, meningitis or tetany. In tetanus the muscular spasm does not fully relax between the attacks.

**Course and Complications.** In fatal cases the spasms become more severe and frequent. Complications include rupture of a muscle and bronchopneumonia.

**Prognosis.** Early and efficient treatment renders the outlook more hopeful, but even then the mortality is usually over 50%. Death may occur in 3 or 4 days from asphyxia or heart failure. The prognosis is usually worse if tetanus rapidly follows the infliction of a wound. Cole calls the "period of onset" the time between the onset of trismus and the first generalised reflex spasms. A period of onset of less than 2 days indicates a very grave prognosis. The prognosis is better with wounds of the lower extremities.

**Treatment.** *Prophylactic.* Tetanus toxoid, 1 mil. given subcutaneously and repeated in six weeks, is used for prophylactic inoculation of

members of the fighting forces. It may be combined with T A B vaccine and given at a month's interval. Antitetanic serum should be given in any case of a wound which may have been contaminated by soil. The prophylactic dosage for an adult is 500 units subcutaneously, repeated three times at weekly intervals. Larger doses are required if there are septic wounds, i.e., 8 000 to 5,000 units every other day for 3 doses, and then weekly injections for 3 weeks or until the wound is healed. The wound should also be disinfected with 1 in 20 carbolic acid solution.

**Curative** The patient should be nursed on a water bed in a dark, quiet room. A cradle should be used to keep the bedclothes away from his body. A single large intravenous injection of 200 000 international units of antitetanic serum should be given. The effect of this will last for 10 days. No advantage is gained by intrathecal or cisternal injection of serum. Some authors recommend in addition the subcutaneous injection of 1 mil of tetanus toxoid, at the first onset of symptoms. This is repeated at intervals of 5 or 6 days. After an interval of at least an hour the wound should be opened, syringed with hydrogen peroxide and a light gauze dressing applied, which will not exclude the air. This dressing is repeated every 4 hours. If there are no reflex spasms sod. brom. gr 30 should be given by mouth every 6 hours. Reflex spasms are best controlled by the rectal injection of Avertin (bromethol B P Add), using 0.1 mil per kg of body weight or of paraldehyde m 60 per stone of bodyweight, in 10% dilution in normal saline. This usually has to be repeated every 6 to 8 hours, until the spasms no longer recur. If the respirations become rapid and shallow, and if there is cyanosis, an injection of atropine sulphate gr 1/120 should be given hypodermically, and warmed oxygen administered through a nasal catheter. If there are severe respiratory spasms before the patient is under the influence of the Avertin they should be controlled by gas and oxygen, or by chloroform. The use of curarine and a Drinker's respirator is not recommended. A special nurse is required day and night to feed the patient. A dietary containing at least 2 000 calories daily is necessary, in the form of dextrose lemonade, eggs and milk. If there is profuse sweating 1 to 2 pints of normal saline should be given by mouth or by rectum. If the patient cannot swallow he should be fed through a nasal tube passed into the stomach which should be left in position between the feeds.

### Actinomycosis

**Definition** Specific granulomatous lesions caused by a mycotic organism.

**Etiology** The cause is the *Actinomycetes bovis* (streptothrix actinomycetes or ray fungus). Wolff and Israel showed this to be a strict anaerobe. The aerobic streptothrix of Bostroem which is found in barley and grasses is probably non pathogenic. The so called actinomycotic lesions in cattle such as 'woody tongue', and 'lumpy' jaw, are usually due to an aerobic organism, the actinobacillus lignieresii, but some of these lesions are due to the actinomycetes bovis. Grains and

grasses are thought to cause abrasions through which the infecting organisms enter. Man is probably infected by the organism entering damaged mucous or cutaneous surfaces. The most frequent portals of entry are the teeth, tonsils, appendix, and the skin. Males are chiefly affected, between the ages of 20 and 40.

**Pathology.** The granulations break down to form abscesses containing little pus, which often discharge through multiple sinuses. Yellowish "sulphur" granules are present in the pus, consisting of a central mycelium and peripheral clubs. The lesions occur most frequently in the head and neck. They may be found in the lungs, pleura or chest wall, in the appendix, cæcum, rectum or liver, in the kidneys or female genital tract, or in the skin. A blood-borne infection may result in cerebral lesions.

**Clinical Findings.** The onset is usually insidious, and the findings differ widely according to the site of the lesion.

**Cervico-facial Type.** This is a surgical condition. Swellings occur in the face, jaw-bone, or neck, which gradually soften with multiple sinus formation.

**Thoracic Type.** This is described on p. 168. The clinical picture in the early stages may resemble that of bronchitis or of pleurisy; later, an empyema, pulmonary tuberculosis, syphilis or a new growth of the lung may be suggested.

**Abdominal Type.** The lesion usually spreads from the appendix to the cæcum. Secondary liver abscess may develop, or an abdominal swelling may form resulting in sinuses through the abdominal wall. It is often only detected at an operation for "appendicitis."

**Genito-urinary Type.** The clinical picture is that of salpingitis or ovaritis, or rarely of a suppurative renal lesion.

**Cutaneous Type.** The skin and subcutaneous tissues alone may be involved. Possibly infection occurs here from straw.

**Nervous Type.** Infection may spread from the naso-pharynx, along the olfactory nerves to the brain, causing an isolated lesion near the pituitary or the fornix. Direct spread may occur from the jaw to the base of the brain, or a cerebral abscess or meningitis may follow a pulmonary lesion.

**Vertebral Type.** The vertebrae may be affected secondarily to a focus elsewhere. Collapse of the body of a vertebra may ensue.

**Differential Diagnosis.** The condition may closely simulate sarcoma, tuberculosis, or a pyogenic infection. The diagnosis is established by bacteriological examination of the pus; a special request should be made to this effect when the material is sent to the laboratory.

**Course and Complications.** The course is a chronic one. Complications are rare, and are due to the infection being blood-borne to other sites, such as the liver and brain.

**Prognosis.** This is favourable in the cutaneous and cervico-facial types, especially if adequate treatment is given early. It is unfavourable in the deep-seated cases.

**Treatment.** The pus should be evacuated surgically if possible. Potassium iodide should be given by mouth in gradually increasing

doses up to gr 90 three times a day. The patient is usually tolerant of large doses. Successful results have been reported in cases of actinomycosis affecting the abdomen, jaw, chest, etc., by the administration of Sulphapyridine (M & B 693) in doses of 1 G t d s for 6 days, followed by a second course 10 days later. Local application of radium or deep X-ray treatment is of value in some cases.

### Glandular Fever

#### *(Infectious Mononucleosis)*

**Definition.** An acute infectious disease characterised by glandular enlargement, fever and excess of mononuclear cells in the blood.

**Etiology.** The cause is unknown. It is probably due to an ultra-microscopic filterable virus. *Predisposing causes* 1 Age. Children and young adults of both sexes are especially affected. 2 Season. It tends to occur in the winter and spring, and there may be small epidemics.

**Pathology.** The glands show lymphoid hyperplasia with loss of their normal structure, changes closely resembling those seen in lymphatic leukaemia.

**Incubation Period.** This is usually 7 to 8 days.

**Clinical Findings.** The patient is usually a child or young adult, who may not feel well for a few days before being definitely taken ill. He then complains of headache, fever, pain in the neck, and perhaps soreness of the throat and vomiting.

**On Examination.** Three types are described—The glandular, the anginose, and the febrile. *The glandular type.* The tongue is furred and the fauces are red. Enlarged glands may be seen and felt in the neck and under and behind the sterno mastoid muscles. These are usually unilateral, but both sides are affected later. Torticollis may be present for a few days. Enlarged glands may also be found in the submaxillary region, in front of the ear, in the axillæ and in the groins. They are firm and a little tender, and the patient usually holds the neck rather stiffly. The temperature is raised to about 103° F or over, and the pulse is proportionately rapid. Enlargement of the bronchial glands may result in a paroxysmal cough, and there may be abdominal pain if the mesenteric glands are affected. The spleen may be felt in about one third of the cases. Jaundice rarely occurs, at times during the course of the disease, less often it is the presenting symptom before there is any apparent glandular enlargement. In the few cases recorded it has usually been obstructive in type, but occasionally the van den Bergh reaction is indirect and bile is present in the feces. In the latter cases the jaundice cannot be due to pressure of enlarged glands in the portal fissure. *The anginose type.* A membrane is seen on or near the tonsils, resembling that of diphtheria. The cervical glands are not as large as in the glandular type. There may be œdema of the neck with considerable local tenderness. The temperature often remains raised for 2 to 3 weeks. *The febrile type.* This affects chiefly adults. A pink maculo-papular rash appears, chiefly on the trunk between the fourth and seventh days. Glandular enlargement is not



usually noted until the end of the third week. Relapses may occur for many months. The blood: White cells. There is a leucocytosis, of about 20,000 per c.mm.; 80 to 90% are mononuclear cells, either immature lymphocytes, large or small lymphocytes, or large mononuclears. The red cells are not usually affected. In some cases there is no leucocytosis, and the mononuclears are not more than about 40%. The blood changes may also be of very short duration. The Wassermann reaction is positive during the second and third weeks in about half the cases. Paul and Bunnell state that the blood serum agglutinates sheep's red cells in a dilution of 1 in 64 by the fourth day of the disease, and that this test is diagnostic. A positive Paul-Bunnell reaction may also be given in serum sickness, but the heterophile antibody present in these cases can be distinguished from that of glandular fever by absorption tests. The urine: Albumin and blood may be present.

**Differential Diagnosis.** Glandular fever is differentiated from mumps, as the parotid gland is not involved. Other conditions which have to be excluded are diphtheria, leukaemia, Hodgkin's disease, and enlarged glands due to tuberculosis, syphilis, German measles, typhoid fever or sepsis. The white cell count and glandular enlargement may suggest acute leukaemia, but the absence of changes in the red cells is of great significance. The cough and blood count may also suggest whooping-cough. Appendicitis may be simulated if the abdominal glands are affected. The course of the disease usually renders the diagnosis clear. Early jaundice and fever may suggest catarrhal jaundice or cholecystitis. In agranulocytic angina there is leucopenia, with over 90% of lymphocytes, and an almost complete absence of granular cells.

**Course and Complications.** The temperature usually falls to normal in 1 to 2 weeks, but may remain slightly raised in the evening for 6 months; the glands may remain palpable for several weeks. A relapse may occur about 8 weeks after the onset, with fever and enlargement of glands. Suppuration is rare. *Hæmorrhagic nephritis* may occur.

**Prognosis.** The disease is rarely fatal, although death may occur from bronchopneumonia in the anginose type.

**Treatment.** The patient should be isolated in bed for 2 weeks; there is no specific treatment. During the acute febrile stage the patient is kept on a fluid diet, and the bowels should be opened daily. During convalescence an iron tonic may be given, such as syr. ferri phosphat. co. m. 60 t.d.s.

### Glanders

(Farcy)

**Definition.** A specific granuloma, caused by a definite bacillus.

**Etiology.** Glanders is caused by the *Pfeifferella mallei* (*B. mallei*). The disease is transmitted to man by direct contact with an infected animal, such as a horse, donkey or mule, the organisms entering an abrasion in the skin or nasal mucous membrane. It may also be conveyed from man to man.

**Pathology.** The bacilli form an endotoxin, mallein. A granuloma results from the local inoculation, and septicæmia or pyæmia may

ensue The nasal lesions constitute glanders, the subcutaneous nodules are called "farcy buds" and the enlarged lymphatics "farcy pipes" Abscesses may be found post mortem in the liver and spleen

**Incubation Period** This varies between 3 days and 3 weeks

**Clinical Findings** *Acute glanders* The patient is usually a worker amongst horses He complains of malaise headache, and at times of nausea and vomiting

**On Examination** The temperature is raised to 101° F or more, and runs an irregular course The initial lesion may be seen on the hand, arm or face as a papule surrounded by a red area The lymphatics may stand out as red streaks, a generalised papular or pustular eruption may then appear and abscesses form subcutaneously or intramuscularly In other cases lesions also occur in the nose and are accompanied by a sticky exudate, ulceration of the nose and enlargement of the cervical glands Pneumonic and rheumatic forms of acute glanders are also described Glanders bacilli may be isolated from the nasal discharge, the abscesses or the sputum *Chronic glanders* This is characterised by the appearance of subcutaneous nodules generally on the forearms which ulcerate and have a purulent discharge Constitutional disturbance is slight

**Differential Diagnosis** The diagnosis is suggested by the patient's occupation The lesions must be differentiated from other granulomata, such as actinomycosis or syphilis, and the generalised pustular eruption, from small pox The bacteriological findings establish the diagnosis

**Course and Complications** Acute glanders is usually fatal in under 2 weeks Chronic cases may persist for months and then gradually abate, or they may suddenly become acute

**Prognosis** The acute variety is practically always fatal, in the chronic form the outlook is much more favourable

**Treatment** *Prophylactic* Animals suffering from glanders should be destroyed

*Curative* There is no specific treatment The patient must be kept in bed and abscesses opened as they form

## Anthrax

(*The Malignant Pustule Wool sorter's Disease*)

**Definition** An acute specific infective disease, which may assume a cutaneous, pulmonary or intestinal form

**Etiology** Anthrax is caused by the *Bacillus anthracis* which forms spores and thus exists for long periods in a viable stage, outside the body Animals such as sheep, cattle, goats horses and reindeer, after eating infected grass, suffer from "splenic fever" Man is infected on the skin (the malignant pustule) by handling infected hides of animals, or by using infected shaving brushes usually made from horse-hair The pulmonary variety of anthrax results from inhaling spores or bacilli in woollen occupations (wool sorter's disease), and possibly the intestinal form is due to eating infected meat

**Pathology** The organisms are often present in the blood stream,

causing a septicæmia; in wool-sorter's disease the bronchial glands are enlarged, subpleural hæmorrhages may be present, and areas of œdema or collapse are seen in the lungs, with bronchitis. In the intestinal variety hæmorrhagic areas may be seen in the mucous membrane of the bowel, with thrombosis of mesenteric veins. Meningitis may be present as a complication in any septicæmic form of the disease.

### The Malignant Pustule

**Incubation Period.** This is usually less than 24 hours.

**Clinical Findings.** Infection occurs through an abrasion in the skin. The patient may be a worker in hides, or be infected through a shaving brush. Itching is first noticed on the skin of the face, arm or neck, and a small red papule then appears at the site of the itching, which quickly becomes inflamed and angry-looking. The patient may feel quite well up to within a few hours of his death, or be very ill with malaise, shivering and headache. He suffers little or no pain in the lesion.

**On Examination:** The papule is seen encircled by whitish vesicles, and in a day or so a black slough forms in the centre. The tissues around become brawny, and the neighbouring glands enlarge. The temperature may be normal or raised to about 103° F. In severe cases the blood culture is positive before death, which occurs in about a week from the onset.

**Differential Diagnosis.** The malignant pustule must be differentiated from a septic spot or boil, a chancre, or a subcutaneous lesion in glanders. The appearances are very suggestive in anthrax, the history is usually confirmatory and anthrax bacilli are found in the serum from the vesicles.

**Prognosis.** The pustule may disappear spontaneously. If the case is treated efficiently within a day or so of its onset the hope of recovery is very good. The average mortality for cutaneous anthrax is 5%.

**Treatment. Prophylactic.** All shaving brushes should be free from anthrax spores.

**Curative.** The pustule should not be excised, as this increases the risk of septicæmia. It should be covered with gauze to absorb exuding serum, and the affected part of the body kept absolutely still by splints or sandbags. Selazo's serum is injected intravenously in doses of 100 mls, followed by 40 mls every 4 hours for 48 hours. An intravenous injection of neoarsphenamine in doses of 0.6 G. for a man, and a smaller dose for a woman or child, may be given, and repeated once after a day's interval as an alternative to, or combined with the serum treatment. Sulphapyridine (M. & B. 693), 2 G., followed by 1 G. 4 hourly for 48 hours, and then 1 G. t.i.d. for 36 to 48 hours, should be given by mouth in addition, if the patient does not immediately respond to the above treatment.

### Anthrax Œdema

A diffuse œdema of a limb, the face or body may occur, without any local pustule being seen. This variety is always fatal.

**Wool-sorter's Disease***(Pulmonary Anthrax)*

**Clinical Findings** The patient is usually suddenly taken ill with malaise and shivering, followed by pain in the chest, cough, expectoration and much weakness

**On Examination** The appearances are those of a very severe illness, the temperature is usually over 103° F, the pulse and respirations are rapid. The lungs show signs of generalised bronchitis. The sputum is frothy and blood stained, and may contain anthrax bacilli. The blood culture is usually positive before death. The disease is usually fatal within a few days.

**Treatment** Selavo's serum should be given intravenously in doses of 100 mls, repeated as above and Sulphapyridine administered by mouth.

**Intestinal Anthrax**

This is a rare variety. The clinical picture resembles that of acute food poisoning, with abdominal pain, vomiting and diarrhoea. The spleen may be palpable, and anthrax bacilli are found in the faeces. It is usually fatal.

**Treatment** Injections of Selavo's serum are given as for the pulmonary form and Sulphapyridine is taken by mouth.

**Hydrophobia***(Rabies La Rage Lyssa)*

**Definition** A disease caused by the bite of certain rabid animals.

**Etiology** The virus is not definitely known, it is probably ultra microscopic and filterable. It is present in the saliva of mad dogs, cats or wolves. Vampire bats were probably the carriers of the virus in an outbreak in Trinidad. Man is infected by a bite, or by the rabid animal licking a raw surface on his body. The disease is practically non-existent in England now, having been eradicated by the orders for the muzzling of dogs, and later by the quarantine laws which are still enforced.

**Pathology** Negri bodies are found in the brain of rabid animals. They occur in nerve cells, especially in the hippocampus major and cerebellar cortex. They are possibly protozoal. It is not known whether they represent a stage in the life history of the causative organism or whether they are a cellular reaction to the virus.

**Incubation Period** The average is 6 weeks, but it may vary from 12 days to 8 months.

**Clinical Findings** The patient may be a child or adult, who gives a history of being bitten by a rabid animal, usually a dog. The bite heals but at the end of the incubation period symptoms are noted. The disease falls into three stages. *The invasion* Irritation or pain is felt at the site of the bite and the patient becomes irritable or depressed and does not feel well. The voice may be a little hoarse. The temperature is found to be slightly raised. *The stage of excitement* The patient is now acutely ill and in a pitiable condition. He is very restless, and

is seized with painful muscular spasms affecting the muscles of deglutition and respiration. The spasms are provoked by swallowing, or even by the sight or thought of food or water, or by any sudden stimulus.

*On Examination:* The temperature is raised to over  $101^{\circ}$  F., and the pulse is frequent. Saliva may run from the mouth, and mucus from the nose; the patient is cyanosed during the spasms, and may be maniacal (*la rage furieuse*). This stage lasts for 2 to 3 days, and then passes into: *The stage of paralysis.* The patient is now exhausted and muscular paralysis develop. The temperature falls, there is unconsciousness, and death from heart failure occurs in a few hours.

*Varieties. La rage muc.* This is rare in man. There is no excitable stage, the patient is quiet and is rapidly paralysed and dies.

*Differential Diagnosis.* The diagnosis is established by the history of the bite of a rabid animal, the typical clinical findings, and examination of the brain of the animal which shows Negri bodies. If the suspected dog is alive 10 days after the bite, the diagnosis of rabies is excluded. Rabies must be differentiated from:—*Pseudohydrophobia:* This is a hysterical condition occurring after a dog bite. There are no true spasms, but the patient may bark or bite like an animal. Tetanus or strychnine poisoning: The history here is suggestive, and the muscles of deglutition and respiration are not involved in the manner typical of hydrophobia. Acute bulbar paralysis: Here there is no history of a bite, and the maniacal symptoms do not occur. Acute poliomyelitis: It may be impossible to differentiate this during life, as in the epidemic in Trinidad in 1931.

*Course and Complications.* The disease is rapidly fatal, lasting 4 to 5 days, and there are no complications.

*Prognosis.* Multiple bites are more serious than solitary ones, and bites on the head and face are followed by a more rapid onset than those occurring distally from the central nervous system. Hydrophobia is more likely to follow bites of wolves than those of dogs. The disease is invariably fatal if not checked during the incubation period.

*Treatment. Prophylactic.* The importance of muzzling dogs in endemic zones and of quarantining imported dogs has been mentioned. A dog bite should be allowed to bleed freely, and then immediately washed with 1 in 1,000 perchloride of mercury solution, and cauterised with fuming nitric acid. The patient should then be sent to a Pasteur Institute for a series of inoculations with material obtained from the spinal cord of rabbits, which have been inoculated intracerebrally with the virus of rabies before being killed. If given early, rabies can be prevented in the vast majority of cases.

*Curative.* Only palliative measures can be employed, such as injections of morphine or inhalations of chloroform.

### Psittacosis

*Definition.* An acute infectious disease conveyed by parrots and allied birds, characterised by fever, prostration, and often by pulmonary symptoms.

*Etiology.* The *Bacterium psittacosis* was isolated from the bone

marrow of affected parrots in 1893. This organism belongs to the *Salmonella* group. It has rarely been recovered from human cases or from parrots subsequently, and it is not now believed that it is the causative agent. It is now generally accepted that the disease is due to a filterable virus. Epidemics occur chiefly in houses and flats. In July 1929 there was a fairly extensive epidemic in the Argentine, and cases occurred in England in the autumn and winter of 1929-1930, probably due to parrots imported from South America. Budgerigars (love birds) may also transmit the disease. In 1934 red cockatoos (galahs) and budgerigars imported from Australia to England were found to be infected. In Germany in 1934 over 150 cases of psittacosis occurred in six months. The source of infection was found to be apparently healthy budgerigars, who are carriers of the virus, the virus probably having been imported into the country by parrots before 1931. The virus is thought to be present in the bird's faeces and beak discharges. Infection of man is probably through the respiratory tract. Direct infection from man to man is uncommon.

**Pathology** The spleen is a little enlarged, red and soft. The lungs. Petechial hæmorrhages may be seen under the pleura, and areas of consolidation occur, which are dull red and dry, with no evidence of suppuration.

**Incubation Period** This is about 10 days.

**Clinical Findings** The patient is usually an adult who gives a history of contact with a parrot or budgerigar which was ill or has subsequently died, or with a budgerigar apparently in good health, which is a carrier of the virus. The onset is somewhat sudden, with malaise, shivering, headache, and at times severe epistaxis, nausea, vomiting or diarrhoea.

**On Examination** At the onset the temperature is raised to about 102° to 104° F, but the pulse is slow, under 100. The patient may be drowsy or complain of severe occipital headache. The temperature remains raised for 1 to 3 weeks, and gradually falls by lysis. A cough often appears after a few days, and examination of the lungs shows signs of bronchitis. This may be followed insidiously by the development of areas of consolidation or collapse in the lungs, which disappear as the temperature falls. In some instances the patient is overwhelmed with toxæmia, he has a low muttering delirium, and a Parkinsonian-like expression is very obvious. In other cases there is much abdominal distention, with offensive loose motions. Small red spots may appear on the chest or back. The spleen is rarely palpable. The urine often contains albumin. The blood. The white cells usually number about 7,000 per cmm, but in some cases a leucocytosis has been recorded. The blood may contain the virus during the first four days of the disease, as shown by mouse inoculation. A complement fixation test, using as an antigen 5% virulent mouse spleen in phosphate or saline is used as a diagnostic test, and a mouse test can also be employed, the animal being infected by the patient's sputum or pleural fluid. A positive agglutination with one of the organisms of the enterica group has been noted at times in the first week of the illness.

**Differential Diagnosis.** The disease is diagnosed chiefly on circumstantial evidence, and must be differentiated from influenza, enterica infections, miliary tuberculosis, pneumonia or *B. coli* urinary infection. The onset is more sudden than in enteric fever. There are neither eye nor limb pains as in influenza. The urine does not contain the colon bacillus. There is no rusty sputum, and the aspect does not suggest pneumonia. The history of contact with a sick parrot or budgerigar, the presence of the typical clinical picture described above, and the demonstration of the virus in the bird or the patient enable the diagnosis to be made.

**Course and Complications.** The average course is 3 weeks. Broncho-pneumonic areas of consolidation may be regarded as complications.

**Prognosis.** The disease is serious, the mortality varying between 10 to 30%. It is worse for old people.

**Treatment. Prophylactic.** It is now illegal to import all birds included under the heading "parrot" except under licence and three months' quarantine should be imposed. The sick bird should be destroyed by coal gas or chloroform and the carcase burned, taking care that the operator wears rubber gloves, which are also burned. The danger from healthy budgerigar carriers still exists in this country.

**Curative.** The patient should be kept in bed until the temperature has been normal for 4 days. There is no specific treatment, and only symptomatic and general measures are available.

### Trench Fever

**Definition.** An acute infectious disease characterised by fever, pains in the shins and muscles, and frequently enlargement of the spleen.

**Etiology.** Trench fever is probably caused by an ultramicroscopic filterable virus. This is present in the blood and urine of patients. It is conveyed by lice, as their faeces contain the virus, which is inoculated into the skin by scratching. Patients' blood may remain infectious for over a year. Trench fever was very prevalent in the 1914-18 war amongst troops infested with lice. It does not occur in England now.

**Pathology.** Trench fever is not a fatal disease, and there is no morbid anatomy.

**Incubation Period.** This is usually between 10 and 24 days.

**Clinical Findings.** The onset is generally sudden with malaise, shivering, giddiness, and pains in the head, behind the eyes, and the back and legs. There may also be nausea, vomiting, sweating and either diarrhoea or constipation. During the acute stages the patient complains of photophobia and general muscular aching, and severe shooting or boring pains are felt in the shin bones, which are worse at night. These shin pains are not present in all cases.

**On Examination:** The patient is usually flushed, the skin moist and the conjunctivæ injected. The shin bones are very tender and the skin over them hyperæsthetic. A rash may be seen on the chest and abdomen consisting of oval or circular dull red macules, about 2 to 10 mm. in diameter, which appear in crops and last about 24 hours. They fade

on pressure. There are several types according to the temperature: (a) A three or five day fever. (b) A spiky fever, the temperature suddenly shooting up to about 103° F. for a few hours, every fifth, seventh or eighth day on 3 or 4 occasions. (c) A prolonged fever lasting 2 or 3 weeks or more. The pulse is usually about 100 or higher during the fever. The spleen is enlarged slightly during the fever, but it may be difficult to feel owing to tenderness of the abdominal muscles. The blood. There may be a leucocytosis of about 20,000 per c mm. The urine. A trace of albumin may be present.

**Differential Diagnosis.** Trench fever must be differentiated from influenza, relapsing fever, malaria or typhoid group infections. The last three are excluded by blood examinations. The temperature curve, shin pains and exposure to lice distinguish it from influenza.

**Course and Complications.** Relapses are prone to occur, with recurrence of fever and pains at long intervals after the primary infection, especially if patients return to duty too soon. Tachycardia is noted as a frequent complication, rarely hæmorrhagic nephritis occurs.

**Prognosis.** The patient is usually fit in about 2 months. Death does not occur.

**Treatment.** *Prophylactic.* Delousing of troops should be carried out at frequent intervals.

*Curative.* All lice must be removed from the patient, he should be kept in bed for a week after the last relapse. Pain may be relieved by drugs, such as aspirin gr 10 t d s. Compresses, rung out in a saturated solution of magnesium sulphate, in some cases relieved the shin pains.

### Coccidiosis

#### (California Disease)

**Etiology.** Coccidiosis is caused by a fungus, the *Oidium coccidiodes*. It occurs in America. Man is usually infected by inhalation, and at times through the skin.

**Pathology.** The fungus produces caseous nodules in the liver, spleen, lungs, bones and skin, and less often in the kidneys, peritoneum and pericardium.

**Clinical Findings.** The patient is usually a young man acutely ill with fever and prostration.

**On Examination.** In pulmonary cases the findings resemble those of pulmonary tuberculosis. Subcutaneous nodules or an abscess discharging through the skin may be present. The liver and spleen may be felt enlarged.

**Differential Diagnosis.** The diagnosis is not usually established until after death, when the causative organism is found in the lesions.

**Course and Complications.** Death usually occurs in 2 weeks in acute cases.

**Prognosis.** The outlook is hopeless.

**Treatment.** There is no curative treatment.



## CHAPTER IX

### THE LOCOMOTOR SYSTEM

#### THE MUSCLES AND FASCIÆ

##### Fibrositis

##### *(Muscular Rheumatism. Myalgia)*

**Definition.** Inflammation of the connective tissue of muscles, fasciæ, ligaments, nerve sheaths, tendons and periosteum.

**Etiology.** This is uncertain. Fibrositis is probably due to bacterial toxæmia, the source of infection being in the intestine, or in other sites such as the teeth, tonsils, sinuses or gall-bladder. Exciting causes are strain and cold.

**Pathology.** Small inflammatory nodules are formed in some cases in the muscles, and inflammatory changes occur in the connective tissues.

**Clinical Findings.** The patient is usually an adult. There may be a history of cold, exposure, or commonly of muscular strain. The lumbar muscles are often affected (lumbago) or those of the neck (stiff neck) or chest (pleurodynia). An attack of lumbago may come on quite suddenly on stooping or on making a violent movement with the leg or arms, such as braking or cranking a car. The patient is then seized with excruciating pain in the small of the back, and feels as if the spine were broken in two. He is fixed in one position, and can only move with assistance. In the sub-acute cases there is a less severe pain felt in the back, which is intensified on stooping, lifting, sneezing or coughing. On deep palpation the nodules, which are very tender, can usually be felt. A stiff neck (torticollis) usually follows sitting in a draught, one or other sternomastoid or trapezius muscle being affected. The intercostal muscles may be involved, causing pleurodynia, which is usually unilateral.

**Course and Complications.** In lumbago the inflammatory process may extend to involve the sheath of the sciatic nerve, with consequent sciatica. Generally the patient recovers from an acute attack in 3 to 4 weeks, but he is liable to recurrences.

**Treatment.** During the acute stage rest and warmth are essential. In acute lumbago an injection of Novocain 2% and adrenaline 2% (injectio procain. et adrenalin.) into the tender nodules, or just beneath the surface of the fascia covering the affected muscle, often gives rapid relief of pain and freedom of movement. 0.5 mil. is injected at each site, up to a maximum of 20 mils. After the injection the patient is instructed to put the affected muscles through a full range of movement, and this should cause no pain. The patient should only remain in bed if the pain is very severe and Antiphlogistine (cataplasma kaolini B.P.) may be applied to the back every 12 hours during the acute stages.

Aspirin gr 10 t i d may be given for the relief of pain, and in very severe cases it may be necessary to inject morphin sulph gr  $\frac{1}{4}$  intra muscularly at the site of the pain. The nodules can often be dispersed by massage. Subsequently any focus of sepsis should be eradicated and care taken that the bowels are completely evacuated daily, as there is often some degree of constipation present. All strains to the back should be avoided and occupations involving stooping are most unsuitable.

### Epidemic Myalgia

#### (*Epidemic Pleurodynia Bornholm Disease*)

**Etiology.** This is a rare disease of epidemic nature (probably a virus infection), occurring chiefly in the summer months and affecting principally children and young adults.

**Clinical Findings.** The incubation period is thought to be 2 to 4 days. The disease is characterised by a sudden onset of pain around the diaphragmatic attachments, often unilateral. Pains may also occur in the abdomen, back, or neck, and frontal headache is a characteristic feature. The temperature is raised to about 104° F for 24 to 48 hours. Dry pleurisy may be present. The disease must be differentiated from an acute abdominal lesion especially when there is vomiting and abdominal distention. It may also resemble influenza but diaphragmatic pleurisy is not a characteristic feature of the latter. There is a tendency for a relapse to occur 2 or 3 days after the temperature has fallen to normal.

**Treatment.** This consists in rest in bed, a bandage round the lower ribs to diminish diaphragmatic movement and aspirin for relief of pain.

### Primary Suppurative Myositis

**Pathology.** A single abscess usually forms in the muscles, less commonly there are multiple foci of suppuration or a diffuse purulent infiltration occurs. The infection is generally staphylococcal.

**Clinical Findings.** Primary suppurative myositis is a rare condition, occurring chiefly in Japan, in field labourers. The onset is usually sudden, with fever, muscular pain, localised tenderness and swelling in a muscle, and œdema and discoloration of the overlying skin. Fluctuation occurs in about 7 to 10 days.

**Course and Complications.** Recurrences or multiple abscesses may occur. Muscular atrophy or contracture may follow.

**Treatment.** The abscess should be opened and drained.

### Dermato myositis

#### (*Non suppurative Myositis*)

**Definition.** A disease characterised by inflammation and degeneration of muscles, subcutaneous œdema and various forms of dermatitis.

**Etiology.** This is unknown, but probably dermato myositis is due to an infection.

**Pathology.** The muscles are œdematous, pale red or yellow. Areas of round celled infiltration are seen in the muscles with atrophy of the

muscle fibres and thinning of the epidermis. Hæmorrhages may occur (*Polymyositis hæmorrhagica*).

**Clinical Findings.** The onset is usually insidious, with pain or cramps in the muscles, anorexia, malaise and pyrexia.

**On Examination:** Oedema of the face and eyelids may give the appearance of alabaster. Generalised oedema may occur. Various erythematous skin rashes may be seen. The spleen is enlarged and various muscles are tender, weak, wasted and often stiff.

**Prognosis.** Death occurs in about 60% of cases, and may result from involvement of the muscles of respiration.

**Treatment.** The patient must be kept in bed; massage and electrical treatment may be used, and the pain relieved by analgesics such as aspirin. The basal metabolic rate should be determined. In some cases it is low and good results are obtained from the administration of thyroideum, starting with gr.  $\frac{1}{4}$  daily and gradually increasing the dose.

### Primary Myositis Fibrosa

**Etiology.** This is unknown.

**Pathology.** The muscle fibres atrophy, and are replaced by connective tissue.

**Clinical Findings.** The patient notices swelling and pain in the muscles of the legs.

**On Examination:** There is little tenderness, but the affected muscles are very hard, and subcutaneous oedema may be found over them.

**Prognosis.** The disease often spreads until nearly all the voluntary muscles are affected.

**Treatment.** Massage and electrical treatment may be tried, and improvement often results.

### Progressive Myositis Ossificans

**Etiology.** The cause is unknown.

**Pathology.** Embryonic connective tissue is first formed; this is converted into fibrous tissue, and later ossification occurs. Deposits of bone are found in the muscles, tendons, ligaments and fasciæ, and there are exostoses. Some cases follow trauma and are preceded by a hæmatoma.

**Clinical Findings.** The patient is usually a young adult; men are attacked more frequently than women. The early symptoms suggest muscular rheumatism, and at the onset there may be slight fever with some redness or swelling of the skin over the affected muscles, usually the back and the neck. Bone formation gradually occurs in the muscles and spreads to the ligaments causing fixation of joints. The muscles of mastication may be involved, with fixation of the jaw. Severe scoliosis may ensue. Exostoses are frequently noted on the humerus, tibia, fibula and ribs. The big toes and thumbs are deformed and small, the interphalangeal joint being ankylosed and the metacarpal or metatarsal bone stunted.

**Prognosis** This is very unfavourable, and death usually occurs from some intercurrent infection after the patient has become bedridden.

**Treatment.** There is no curative treatment, good nursing care is required in advanced cases.

Other affections of voluntary muscles which are mentioned under their particular sections include Secondary suppurative myositis (see pyæmia, p 576) Trichiniasis (see p 721) Degenerations (such as Zencker's degeneration see enteric fever, p 554) Hæmorrhage, as in scurvy or in influenza (see p 581), into the rectus abdominalis Rupture, as in tetanus Tuberculosis A cold abscess in muscle may be due to direct spread from a bone abscess, rarely in miliary tuberculosis foci are found in the voluntary muscles In syphilis a gumma may form

### The Muscular Dystrophies

*(The myopathies and muscular diseases of doubtful nature)*

In this group of cases the primary changes are regarded as muscular rather than nervous The characteristic features of the myopathies are — A familial history The age of onset Different muscle groups are affected in different types Pseudo hypertrophy occurs in some cases There are no fibrillary contractions such as occur in cord lesions There is no reaction of degeneration and no sensory changes occur, as in neuritis

#### Pseudo-hypertrophic Muscular Dystrophy

**Etiology.** The cause is unknown The disease tends to run in families, being transmitted by the mother Boys are usually affected

**Pathology** There is atrophy of the muscle fibres, with increase in the fat and connective tissue of the affected voluntary muscles, causing apparent enlargement There are no changes in the central nervous system

**Clinical Findings** The patient is usually a boy, aged 3 to 12 years There is a history that he began to walk late, and that he has been unsteady on his legs and finds stairs difficult

**On Examination** The general condition is good and there is enlargement of the muscles of the calves (gastrocnemius and soleus), the front of the thighs (quadriceps extensor and sartorius), the buttocks (glutei), the infraspinatus, triceps and at times the deltoid The enlarged muscles are found to be weak The patient stands on a wide base, lordosis is marked, the gait is waddling, and in order to rise from a supine posture the patient "climbs up himself," first rolling over on to his hands and knees, and then working his hands up his legs There is usually wasting of the latissimus dorsi and lower part of the pectoralis major muscles, so that if the child is lifted up under the arms he tends to slip through the hands The face, with the exception of the masseter muscles and the forearms are not affected There are no sensory changes The deep reflexes in connection with the affected muscle groups are gradually diminished and there are no fibrillary tremors Electrical reaction. The reaction of degeneration is not

present, but the response to faradisation and galvanism gradually diminishes.

**Course and Complications.** The disease is progressive, and death usually occurs before adult age from wasting or secondary infections.

**Treatment.** There is no curative treatment, but the prolonged administration of vitamin E in the form of fresh dried whole-wheat germ, oz.  $\frac{1}{2}$  daily is worthy of a trial. This may be combined with the subcutaneous injection of vitamin B<sub>6</sub>, the initial dose being mg. 50, followed by mg. 100 to 500 at weekly intervals. Massage and exercises are usually beneficial.

### Juvenile Muscular Dystrophy (*Erb's Dystrophy*)

**Etiology.** The cause is unknown.

**Pathology.** There is wasting of the fibres of the affected muscles.

**Clinical Findings.** The patient is usually between the ages of 15 and 35, male or female, and several members of a family may be affected. Weakness is first noticed in the arms or legs and the muscles are later found to be wasted.

**On Examination :** The muscles wasted are usually those of the upper arm (biceps, triceps and supinator longus) and the thigh (glutei, extensors and hamstrings), giving a bottle-shaped appearance to the limbs. Some of the trunk muscles may also waste, such as the latissimus dorsi, lower part of the pectoralis major, trapezius, rhomboids, serratus magnus, erector spinæ, etc. The face is not usually affected. Lordosis is present, the electrical reactions and deep reflexes are diminished. The reaction of degeneration is not present and there are no sensory changes and no fibrillary tremors. In some instances a distal type (of Gowers and Spiller) may be seen, in which the forearms, hands and wrists, legs, feet and ankles, and muscles of the face are wasted.

**Course and Complications.** The disease is usually progressive, but the patient may live until middle age before being carried off by some intercurrent infection.

### Facio-Scapulo-Humeral Dystrophy (*Landouzy-Dejerine's Dystrophy*)

This is a variation of the juvenile type, which occurs usually in infancy, has the same hereditary factor and affects the sexes equally.

**Clinical Findings.** Weakness and wasting are first noticed in certain face muscles. These are the orbicularis oris and orbicularis palpebrarum; the eyelids cannot be closed and the lips are everted; the lower lip projects (tapir mouth). On smiling the lips are straight. The shoulder girdle muscles, including the trapezius, latissimus dorsi, serratus magnus, pectorals, triceps and biceps, are subsequently affected. The scapulae become winged.

**Course and Complications.** The course is progressive, but the patient may live until adult life.

### Amyotonia Congenita or Myatonia Congenita (Oppenheim's Disease)

**Etiology** This is unknown. The disease resembles a myopathy in many respects.

**Pathology** The anterior horn cells of the spinal cord are usually diminished in number, especially in the lumbar region, and the corresponding anterior nerve roots are thin and deficient in myelin. The muscle fibres on the whole are small, but a few abnormally large ones are found. The connective tissue and fat of the muscle are increased.

**Clinical Findings** The disease may have a familial incidence. The onset is at, or before birth, and the condition is characterised by extreme flaccidity of the voluntary muscles; an infant, if sitting, tends to fall forwards and resembles a frog (Batten's frog child). The muscles are very weak and the child may be unable to hold up its head. The legs are more severely affected than the arms. The hands and feet are long and narrow. The face is not affected. The deep reflexes are absent, and the electrical response to faradisation is diminished, but the reaction of degeneration is not present.

**Differential Diagnosis** Amyotonia congenita is differentiated from poliomyelitis by the general distribution of the paresis and absence of complete paralysis in amyotonia congenita, and from the myopathies by the absence of marked wasting and the tendency to improvement. Its resemblance to the Werdnig Hoffmann disease has been noted on p. 422.

**Course and Complications** The child may soon die from intercurrent infection, but there is a tendency to improvement with an increase of muscle tone and recovery of the deep reflexes, so that he may survive and reach adult life.

### Myotonia Congenita (Thomsen's Disease)

**Etiology** The cause is unknown.

**Pathology** The voluntary muscle fibres may be increased in width. There is no increase in the muscular connective tissue and there are no nervous changes. The muscular contractions resemble those produced experimentally with veratrin, there being delay in contraction and relaxation. Experimental evidence on goats suffering from a similar disease suggests that the lesion is a primary muscular one and not due to changes in the myoneural junctions.

**Clinical Findings** The disease is very rare, occurring more commonly in males, tending to run in families, and first showing itself in childhood. The patient notices a stiffness in the voluntary muscles, chiefly in the hands, arms, feet and legs, and to a lesser degree in the trunk. The muscles of mastication may also be affected.

**On Examination** It is found that the voluntary contraction and relaxation of the muscles is prolonged, but a movement becomes more speedy after repetition. This can be well tested by asking the patient

to grip the hand several times. There is no spasticity found on passive movement. The skeletal muscles are usually enlarged. The sensation is normal. The following distinctions exist between this condition and the myopathies: The deep reflexes are not affected; there is no atrophy of muscles; the electrical reaction is peculiar, the faradic and galvanic responses are first small and prolonged, but on repeated stimulation they become normal (myotonic reaction); there is a modified reaction of degeneration, as A.C.C. approximates to K.C.C. (normally  $K.C.C. > A.C.C.$  and in the reaction of degeneration  $A.C.C. > K.C.C.$ ) (see p. 287).

**Course and Complications.** The disease has no effect on the duration of life.

**Treatment.** Quinine sulphate or hydrochloride gr. 5 to 10 once to thrice daily, should be given by mouth, the dose being increased until the myotonia is relieved. An adequate maintenance dose must be worked out.

### Myotonia Atrophica (*Dystrophia Myotonica*)

**Etiology.** The cause is unknown.

**Pathology.** There is atrophy of the majority of fibres in the affected muscles, with some giant fibres.

**Clinical Findings.** The patient is usually a male, aged between 20 and 35. There is often a family history of the disease and also of cataract. The onset is insidious, with pains in the arms or legs, weakness, loss of weight and a difficulty in relaxing the grip.

**On Examination:** There is wasting of the muscles of the face, the neck (sterno-mastoids), the forearms and the legs. The deep reflexes in connection with the affected muscle groups are diminished. There are no sensory changes, no fibrillary contractions and no reaction of degeneration. Electrical stimulation causes a prolonged relaxation after contraction.

**Course and Complications.** The course, if untreated, is slowly progressive. Death usually occurs from intercurrent disease.

**Treatment.** Quinine should be given as in myotonia congenita (see above).

### Myasthenia Gravis

**Etiology.** The cause is unknown. The disease appears to be due to a chemical abnormality (deficiency of acetyl-choline) which results in a defect of transmission of the impulse from the nerve to the muscle. Acetyl-choline is the chemical substance which allows transmission of impulses across the myo-neural junctions in striated muscles, but acetyl-choline itself has no beneficial effect in the disease. It is thought that eserine and Prostigmin inhibit the destruction of acetyl-choline by an esterase.

**Pathology.** "Lymphorrhages" (small round cells) are found between the muscle fibres. There are no primary changes in the nervous system.

**Clinical Findings** The disease affects men and women equally and usually begins in adults before middle age. The patient may complain of inability to keep his eyes open or of diplopia, which increases during the day and disappears temporarily after a night's rest. In other cases varying fatigue is noticed in different muscles causing difficulty in speech or swallowing, alteration in voice or weakness of the neck or limbs. Thus if the arm is affected, the performance of any movement involving its use will cause rapid fatigue, power being regained temporarily after a short rest. Aching in the limbs may also be noticed.

**On Examination** In a typical case certain muscles supplied by the cranial nerves are affected, especially those of the eyelids and the external oculo-motor muscles, with resulting ptosis, strabismus and inability to close the eyes tightly. The lips may also be involved so that the patient cannot whistle or smile normally. If the palate is affected it rapidly fatigues when the patient is asked to say 'Ah'. The voice may be nasal and nasal regurgitation of fluids occurs on swallowing. The tongue and jaw muscles may also be involved with dysarthria and dropping of the jaw. In other cases there is weakness of the neck so that the head is supported on the hands or fatigability of the arms may be demonstrated for when the patient holds the arms extended they gradually droop to the sides. The muscles are not usually wasted, the deep reflexes are variable, the jaw-jerk may be abolished but the knee jerk is not lost. The electrical tests show the 'myasthenic reaction', the response to faradisation is rapidly exhausted but is restored by rest. Galvanic stimulation does not fatigue the muscles and they will contract voluntarily when they do not respond to faradisation. Sensation is usually unaffected. The urine. The creatine content is very low.

**Differential Diagnosis** The characteristic expression of the patient with drooping eyelids and elevated head and the fatigability of the muscles are typical. Other causes of ptosis must be excluded such as hysteria, tabes dorsalis and cerebral lesions. Organic bulbar paralysis and diphtheritic neuritis must be differentiated in some cases.

**Course and Complications** The course is progressive, but may be interrupted by remissions, especially during pregnancy. Involvement of the respiratory muscles may cause death.

**Treatment** This consists in rest for the muscles and tonics. Electrical treatment should not be given. Beneficial results were obtained in 1929 with ephedrine hydrochloride in doses of gr  $\frac{1}{2}$  to 1 t.i.d., and in 1932 with ephedrine gr  $\frac{3}{4}$  together with glycine (glycocol) oz  $\frac{1}{2}$  twice daily. In 1934 a further advance was made by using Prostigmin, a synthetic physostigmine. If Prostigmin 1 to 2.5 mg and atropin sulph. gr 1/100 are injected subcutaneously the patient recovers power in the affected muscles in about 10 minutes, and the effects are maintained for about 3 to 6 hours. To produce comparable results by oral administration, 50 mg of Prostigmin are equivalent to an injection of 1 mg. A severe case may require as many as twenty 15 mg



tablets of Prostigmin, spaced out through the 24 hours. In each case the maintenance dose must be worked out. To prevent abdominal pains, tnc. belladon. m. 20 should be taken as required. An acute case has been successfully treated by daily injections of desoxycorticosterone acetate mg. 10, followed by the subcutaneous implantation of 3 pellets of mg. 150 each (see p. 666).

### Familial Periodic Paralysis (Intermittent Myoplegia)

**Etiology.** The cause is unknown. The disease tends to run in families.

**Pathogenesis.** The paralysis appears to be connected with the amount and form of potassium salts in the muscle fibres. It has been shown that an attack can be produced in a susceptible individual by the administration of dextrose, G. 200, by mouth. This results in the fall of the blood potassium, paralysis usually appearing when the figure is below 10 to 12 mg. per 100 c.c. (normal 16 to 20 mg. per 100 c.c.).

**Clinical Findings.** The disease usually first shows itself in childhood or about the age of puberty. There are recurring attacks of paralysis which may affect the arms, legs, trunk and neck. Usually the face, eyes, sphincters and diaphragm are not involved. In some cases only the lower limbs are affected and the weakness may be greater in one half of the body than the other. Premonitory symptoms are sometimes noted, such as lassitude, tingling in a limb, hunger, thirst, palpitations or sweating. A heavy meal may predispose to an attack. The onset usually occurs during sleep, the patient on waking finds that he cannot move his limbs. The attack usually passes off in a few hours or in a day or so.

**On Examination:** The affected muscles are flaccid and completely or partially paralysed. The cutaneous and deep reflexes are diminished or abolished. The electrical reactions are diminished or absent. Sensation is usually normal. The blood potassium falls during an attack.

**Differential Diagnosis.** Owing to the rarity of the condition and the transitory nature of the attacks, the patient runs a risk of being diagnosed as suffering from hysteria or malingering.

**Prognosis.** The attacks tend to become less severe and frequent after middle age.

**Treatment.** The attack can usually be relieved by administering at the onset pot. chlorid. gr. 90 to 180 for an adult, and gr. 15 to 30 for an infant, dissolved in 2 oz. of water and taken in a small quantity of milk.

### THE BONES Osteitis Deformans (Paget's Disease)

**Definition.** A chronic disease of bone, characterised by enlargement of the skull, kyphosis and bowing of the extremities.

**Etiology.** The cause is unknown. The disease is almost always associated with atheroma.

**Pathology** The pelvis, spine, skull, tibia, or clavicle is generally first affected. The changes are those of subperiosteal new bone formation and deeper areas of rarefaction. There is much bony thickening and irregularity. The bone fat is increased and its calcium diminished.

**Clinical Findings** The patient is usually a male, aged 40 to 60, and the disease tends to run in families. The onset is insidious, pains may be first noticed in the legs (shins) or enlargement of the head or bending of the bones may be the first symptom. *Examination of a Developed Case* The patient presents a typical appearance, the calvarium is enlarged, and the face appears triangular with the base upward. The legs and arms are bowed forwards and outwards. Kyphosis is marked usually in the upper dorsal region, and the spine is very rigid. There is also thickening and enlargement of the pelvis, shoulder girdle, tibiae and femurs and to a lesser degree of the bones in the arms. The patient's height may be reduced by 4 to 6 inches but the hands and feet are usually unaffected. In rare cases the changes are limited to a single bone. An x-ray examination of the bones shows the enlargement and deformity, and areas of rarefaction and of dense bone are seen, giving a woolly appearance to the skull. The blood. The serum calcium and phosphorus are normal. The phosphatase is increased. *The sedimentation rate of the red cells is likely to be normal.* The urine calcium content is usually increased in the early stages later it may be normal or diminished.

**Differential Diagnosis** There is usually no difficulty in distinguishing Paget's disease from acromegaly, rickets and localised over growth of bone due to other causes, such as trauma or syphilis. The x-ray findings are characteristic. Secondary carcinoma of bone may give rise to difficulty, but the primary growth can usually be found and the x-ray appearances differ. The sedimentation of the red cells is increased in carcinoma.

**Course and Complications** The course is progressive, the patient may live for 20 to 30 years after the onset, gradually becoming more crippled, and dying from an intercurrent disease. Fracture of the long bones, compression paraplegia or osteo sarcoma may occur.

**Treatment** There is no specific treatment. Osteotomy is not usually advisable. Analgesic drugs, such as aspirin, may be required for the pain. In the later stages, when local bony retention of calcium and phosphorus is occurring, treatment with acid and phosphat *gr 30 t i d* may prevent further accumulation in the bone.

### Generalised Osteitis Fibrosa

(of von Recklinghausen *Diffuse Fibrocystic Disease of Bone*)

**Definition** A disease characterised by softening of bones, formation of fibrous tissue and benign giant celled tumours and cysts.

**Etiology** The disease is due to hyperparathyroidism.

**Pathology** The bones are depleted of calcium. The giant celled tumours consist of osteoclastic cells. Some fibrosis occurs in the subperiosteum, bone and bone marrow, and there is local formation

of new bone. A parathyroid tumour is present, usually an adenoma of the principal cells. Rarely there is a generalised hyperplasia of all the parathyroids. Calcification may occur in the kidneys.

**Clinical Findings.** The patient is usually over the age of 20, of either sex, more often a woman. She complains of pains in the back, pelvis or limbs. Spontaneous fracture of a bone may occur. There is tenderness on pressure over the affected bones. The blood: The serum calcium may be as high as 23 mg. per 100 c.c. (normal 9 to 11 mg.), and the phosphorus as low as 1 mg. per 100 c.c. (normal 2.5 mg.). The phosphatase is increased (normal 3 to 13 units, see p. 619). The urine contains an excess of calcium. The calcium in the faeces is usually about normal. In some cases a parathyroid tumour may be felt, or it may only be discovered by an exploratory operation. X-ray examination shows the characteristic bony changes, diminished density and pale areas due to cysts.

**Differential Diagnosis.** The case may be mistaken for one of rheumatism, arthritis or osteomalacia. With secondary malignant deposits in bone, the blood calcium may rise but the phosphorus then rises too, and the phosphatase is normal. In some cases renal symptoms predominate, either due to the formation of calculi or to renal insufficiency. Frequency of micturition may suggest diabetes insipidus. The diagnosis is established by the X-ray and blood changes and by finding a parathyroid tumour, the removal of which arrests or cures the disease. Fragility of bones also occurs as a familial disease associated with blue sclerotics and progressive deafness. The blueness of the sclerotics is due to the dark pigment of the choroid showing through the unduly transparent sclerotics. The deafness results from otosclerosis. The disease usually shows itself in the second or third decade.

**Course and Complications.** The course is usually progressive, unless adequately treated, death occurring from an intercurrent disease such as a pulmonary embolus or from exhaustion.

**Prognosis.** This is good if the parathyroid tumour is removed early. In long-standing cases, no increase in the density of the bones may occur for several years, if at all, although the progress of the disease is arrested.

**Treatment.** An exploratory operation should be made in the neck, and a parathyroid tumour searched for and removed. The tumour may lie in an abnormal site, behind the oesophagus, in front of the second and third thoracic vertebrae, or in the thorax. Subsequently, the fall in the blood calcium is controlled by giving a diet rich in calcium and the administration of vitamin D in the form of liq. calciferol. (B.P. Add.) m. 20 (4,000 i.u.) t.i.d. if there are symptoms of tetany (see p. 660).

### Focal Osteitis Fibrosa (*Osteitis Fibrosa Disseminata*)

This is a condition resembling the general disease, but affecting only one or a few bones, with a definite tendency to spontaneous arrest. It occurs chiefly in adolescents, and there may be spontaneous fracture.

Pigmentation may occur on one side of the body, and premature sexual development may have been noted earlier in life. The blood calcium and plasma phosphatase are normal and the disease is not associated with a parathyroid tumour or with hyperparathyroidism.

### Leontiasis Ossea

A condition of hyperostosis of all the bones of the skull, including the face bones. The cause is not known, but in some cases it may be associated with chronic sinusitis. It occurs usually about the age of puberty and is more common in women than in men. The patient complains of pressure effects, such as headache, neuralgia, deafness, blindness and insomnia. Death may not occur for 30 to 40 years and may be due to intercurrent diseases or to a convulsion.

### Osteomalacia

**Definition** A deficiency disease characterised by softening of bones with a liability to fracture.

**Etiology** Osteomalacia may be due to several causes. 1. Deficiency of calcium and vitamin D in the diet and absence of sunlight. The blood calcium is low and there is a negative calcium balance. It appears to be an adult variety of rickets. This is the cause in the majority of cases. 2. A deficiency of ovarian secretion. This type occurs in association with puberty and pregnancy, probably owing to an excessive loss of calcium. 3. Hyperparathyroidism. In some cases the blood calcium is high and a parathyroid tumour is present. *Osteomalacia is endemic in Northern India, Japan and Northern China.* Males are occasionally affected.

**Pathology.** The bones are brittle, owing to lack of calcium. The bones especially affected are those of the pelvis, spine, thorax and the long bones.

**Clinical Findings** The patient is usually a woman between the ages of 20 and 30, who complains of weakness and aching in the back, chest or legs. Deformity is usually produced, especially in the pelvis, which interferes with childbirth, there may also be kyphoscoliosis, chest deformities, coxa vara and bending of the long bones. A spontaneous fracture of a long bone may occur. The X-rays show that the bones are rarefied.

**Differential Diagnosis** Osteomalacia is characterised by the bony changes in the pelvis, and is to be differentiated from osteogenesis imperfecta, generalised osteitis fibrosa and osteitis deformans.

**Course and Complications** Tetany may occur as a complication.

**Prognosis** A complete cure can usually be effected with adequate treatment.

**Treatment.** The patient should be treated as for rickets with cod liver oil, Halverol or calciferol, sunlight and a diet rich in calcium and phosphorus such as milk, eggs, fish and green vegetables. If the blood calcium is raised and there is no response to antirachitic treatment, an operation should be performed to determine whether a parathyroid

tumour is present, and if found it should be removed. Ovariectomy is not required.

### Multiple Myelomatosis

(*Kahler's Disease*)

This disease is characterised by the formation of reddish tumours in the bone marrow, anæmia and Bence-Jones' proteose in the urine (see p. 442). Spontaneous fractures may occur. The tumours are composed of hæmopoietic cells. The patient complains of aches or pains in the pelvis or chest, and there may be paraplegia. The blood shows a leuco-erythroblastic anæmia, *i.e.*, a hypochromic anæmia with normoblasts. The total number of leucocytes is not increased, but a few myelocytes and myeloblasts are present. The serum globulin is usually increased, and the sedimentation rate of the red cells is rapid. The differential diagnosis includes Paget's disease, Ewing's sarcoma, secondary malignant deposits in bone and syphilis of the bones. The X-ray appearances of the bones, the blood count, the presence of Bence-Jones' proteose in the urine and the result of sternal puncture which shows plasma cells in the bone marrow, serve to establish the diagnosis. The prognosis is hopeless, but X-ray treatment to the bones may relieve the pains.

### Diffuse Osteosclerosis

(*Albers-Schönberg Disease. Marble-bone Disease*)

The bones become unduly rigid owing to thickening with loss of the marrow cavity. The ossification probably starts before birth and fractures are not uncommon. The liver and spleen are enlarged. The blood may show a leuco-erythroblastic anæmia.

### Osteogenesis Imperfecta

(*Fragilitas Ossium*)

A congenital disease characterised by extreme brittleness of the bones, multiple fractures occurring before or after birth. It is sometimes associated with otosclerosis and blue sclerotics. The cause is unknown. The serum calcium, plasma phosphate and calcium excretion are usually normal.

### Osteopsathyrosis

(*Lobstein's Disease*)

This closely resembles osteogenesis imperfecta, but the fragility of the bones is not noticed until childhood or later. It often runs in families.

### Achondroplasia

A congenital disease, characterised by arrested development of the bones of the extremities, with consequent dwarfism. The head is large, the bridge of the nose depressed, and the hands are small with fingers of equal length. Lordosis is present with contraction of the pelvis. The

skin over the body is thick, the voluntary muscles are powerful and the mentality is normal. Males and females are equally affected

### Oxycephaly

This rare congenital disease is characterised by deformity of the skull the vertex being pointed and forehead sloping, together with the occurrence of exophthalmos and optic atrophy. It is considered to be due to premature union of the sagittal and coronal sutures. "Digital impressions" formed by bony trabeculae are seen post mortem on the inner aspect of the vault of the skull, which during life give a beaten silver appearance on X ray examination. The patient complains of headaches, failing vision and possibly loss of smell. Decompression is required to relieve the pressure.

### Hypertrophic Osteoarthropathy

A condition of "clubbing" of the fingers (Hippocratic fingers) or toes often associated with enlargement of the wrists or ankles.

**Etiology.** The cause is not known, impeded venous return and toxæmia may be factors. It is usually found associated with chronic diseases of the lungs and pleura, such as fibroid tuberculosis, bronchiectasis and empyema with mediastinal tumours and congenital disease of the heart. In some instances no cause can be found. It may develop in a few weeks in such conditions as an empyema. Unilateral clubbing may occur (see p. 257).

**Pathology.** Clinically there are two varieties, the drum stick, with bulbous extremities to the fingers, associated with bronchiectasis, and the parrot bill or puffin beak variety, in which the nails are curved from base to tip. This is often met with in tuberculosis. In the early stage the first sign is congestion of the finger tips, at the roots of the nails. In more advanced cases there is enlargement of the hands and feet and swelling of the wrists and ankles, and an intermittent hydrarthrosis may occur in other joints such as the knee. The changes are chiefly in the soft tissues, and by X-rays little alteration is seen.

## THE JOINTS

### Rheumatoid Arthritis

#### (Atrophic Arthritis Infective Polyarthritits)

**Definition.** Inflammatory and degenerative joint changes of doubtful etiology.

**Etiology.** Two types are described, primary or idiopathic and infective. The cause of the primary variety is unknown. The infective type is thought to be due to toxins of organisms especially of the streptococcus viridans or of the Bacterium commune (*B. coli*). The foci of infection may be at the roots of teeth in the nose, throat, cranial sinuses, intestine or genito urinary tract. It has also been suggested

that the joint changes are allergic in nature, or due to a virus. *Predisposing causes* : 1. Age : Chiefly between 20 and 45. 2. Sex : Females predominate. 3. Exposure to cold and damp : The disease is practically unknown in the tropics. 4. Fatigue, overwork and mental strain. 5. Dietetic and possibly endocrine deficiencies.

*Pathology.* The small joints of the hands are especially affected. There is periarticular swelling, and in the early stages excess of fluid in the joints, hyperæmia, lymphocytic infiltration of the joint tissues, and later overgrowth of the synovial membrane with villous projections. Decalcification of bone may be followed by destruction of cartilage, and later dislocation with bony ankylosis may ensue. A rapid atrophy of the small muscles of the hands, and of the larger muscles, such as the extensors of the knee and wrist may occur when the fingers, knees or wrists are affected. Fibrositic changes are also common in the neighbouring muscles.

*Clinical Findings.* It should be realised that the disease is a general one with local joint manifestations. There is usually a prodromal period of fatigue, often accompanied by loss of weight. The patient then notices pain and stiffness, especially on waking in the morning, around a metacarpo-phalangeal or interphalangeal joint. This is followed by increased pain and swelling of the affected joint or joints. The hands and feet are often cold. Later the wrists, elbows, knees, ankles, cervical spine, shoulders, temporo-mandibular or other joints are affected. A periodicity of a few weeks is noticed in some cases between the appearances of fresh lesions. During the early stages acute exacerbations may occur from time to time, characterised by a throbbing pain disturbing sleep. The pain begins suddenly and the patient finds a small red thickening in the skin at the site of the pain, usually over an interphalangeal joint. The patient is very much disabled if the wrists and hands are affected and cannot exert any power in carrying, pulling or gripping, partly owing to pain but chiefly because of muscle weakness.

*On Examination :* In the early stages a spindle-shaped deformity is seen, usually of the proximal interphalangeal joints, with some swelling over the knuckles. Small fibrous nodules may be felt subcutaneously on the palmar or lateral aspects of the phalanges. Subcutaneous nodules may also be felt especially on the dorsum of the forearm. Later, if adequate precautions have not been taken, much deformity may develop in the hands, with ulnar deviation at the wrist and metacarpophalangeal joints, and in advanced cases inability to move or use the fingers or thumbs (see Fig. 60). The tissues around the wrists or other large joints, such as the ankles and elbows, may be thickened and pit on pressure with limitation of movement. There is usually marked wasting of adjacent muscles as described above. The skin of the fingers becomes thin, smooth and glossy. In the infective type the joint lesions may be complicated by tenosynovitis and bursitis, especially around the shoulders, elbows, wrists, knees and ankles. Fixation of the elbows and marked deformity of the hands may prevent the patient from feeding or attending to herself, or lesser degrees of crippleddom may exist.



FIG 60 ULNAR DEVIATION OF HAND IN RHEUMATOID ARTHRITIS

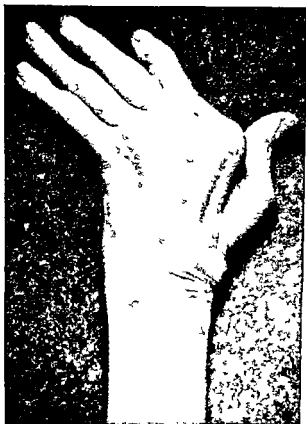


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**FIG 60**    **ULNAR DEVIATION OF HAND IN RHEUMATOID**  
**ARTHRITIS**

painless movement, but no force should be used, otherwise increased pain and swelling will result. Later, various local treatments may be ordered such as active and passive movements, gentle massage, radiant heat, paraffin wax or contrast baths. Aspirin is most valuable for the relief of pain and should be given regularly during the acute stages. By mouth such preparations as liq. iodi simplex may be given in doses of 1 drop in 1 oz. of milk t.i.d. p.c., increasing the dose by 1 drop daily until 30 drops t.i.d. are taken, and then working down the scale again, for a course. Other drugs which may be used include pot. iod. gr. 3 and guaiacol carbonat. gr. 5 in a cachet, t.i.d. Dilute hydrochloric acid in doses of m. 40 to 60, well diluted, should be given after meals if achlorhydria is present. Treatment by protein shock produces improvement in some chronic cases, but it must never be used during the acute stages of the disease. It can be effected by intravenous injection of T.A.B. vaccine, beginning with 25 millions of typhoid and 12.5 millions of Para. A and B bacilli, and working up to 100 or 200 millions in all, at intervals of 5 to 7 days. Usually there is a marked general reaction, the temperature rising to 103° F. or to 105° F. with a focal reaction in the joints. Opinions differ as regards the advisability of using gold salts. Good results are claimed in about 50% of cases treated in Great Britain and in about 33% in America. The treatment is certainly dangerous and its risks should be explained to the patient before it is initiated. A preparation such as Allochrysine or Solganal B is injected intramuscularly at weekly intervals, beginning with 0.01 G. and increasing to 0.1 G., until a total of 1 G. has been injected. A second and third course may be given after a rest period of 3 months. Severe and fatal reactions may occur, especially exfoliative dermatitis, agranulocytosis, and purpura. At the slightest sign of a skin eruption no more gold should be injected. Renal and hepatic disease and severe debility are contra-indications. Gold should only be given if the sedimentation rate of the red cells is over 25 mm. (Westergren) at one hour, the urine free from albumin and the white cell count normal. A complete blood count and urine examination should be made weekly and a sedimentation test monthly. Gold should not be continued if the sedimentation rate is less than 25 mm. (Westergren) at 1 hour. If the eosinophil count rises over 5% the next dose of gold should be halved. If there is albuminuria no further injections should be given. Unless these precautions can be taken gold should not be used. For the decalcification of bone 10 mils of calcium gluconate (B.P. Add.) should be injected intramuscularly weekly. A high calorie diet of mixed type is generally required, but the patient should not be allowed to become overweight. Vitamins B<sub>1</sub> and C should be given in the form of Benerva tab. (Aneurin. hydrochlor. B.P. Add.) mg. 3, 4 daily, and ascorbic acid tab. (B.P. Add.) mg. 50, 2 daily. Orthopaedic treatment may be required to correct deformities in cases inadequately treated during the early stages. The patient should not be allowed to return to work until arrest has been secured as judged by a normal temperature and sedimentation rate, a normal body weight, absence of swelling and stiffness and comparative freedom from pain in the affected joints.

## Osteoarthritis

*(Hypertrophic Arthritis Arthritis Deformans)*

**Definition** Degenerative joint lesions of doubtful etiology

**Etiology** Trauma and strain are exciting factors in some cases

**Predisposing causes** 1 Age Usually over 40 2 Sex Males predominate 3 Metabolic errors The nature of these is uncertain 4 Obesity

**Pathology** The joints most commonly affected are the terminal interphalangeal joints where Heberden's nodes occur, the lumbar vertebrae the knees the sacro iliac joints the lower cervical vertebrae the hips and shoulders There is degeneration of the articular cartilage, with hypertrophy of the surrounding cartilage and bone so that the articular surfaces are approximated The synoviae are usually unaffected Separated osteophytes may be loose in the joints The joint capsule is fibrosed Osteoarthritic changes are present in nearly everyone after the age of 50 but symptoms only occur in a small percentage of individuals

**Clinical Findings** The onset is usually insidious with stiffness and later pain in the affected joints There is no constitutional disturbance except in the rare acute type of disease When the hip is affected the pain may be referred to the front or back of the leg (crural or sciatica neuralgia) or to the knee

**On Examination** The general nutrition is good The affected joint shows some bony thickening and lipping of the bony edges may be felt Creaking is often audible and palpable on movement, and the range of movement is limited. Some muscular wasting may occur above or below the joint and in cases in which the hip joint is affected the patient may walk on his toes with tilting of the pelvis and scoliosis In some instances the disease has an acute onset closely resembling that of acute rheumatic fever The sedimentation rate of the red cells is rarely increased The B M R is below normal in about 30% of cases Radio graphic examination reveals cyst like structures in the bones on the joint surface which result in erosion of the joint cartilage with secondary osteophytic outgrowths

**Course and Complications** Several joints may be affected and one or more of them may be completely immobilised The smaller joints of the fingers may be involved, especially in middle-aged women with development of Heberden's nodes at the proximal ends of the distal phalanges At the climacteric osteoarthritis may occur in the knees associated with obesity

**Differential Diagnosis** This is as for rheumatoid arthritis (see p 611) Limitation of external rotation and abduction of the hip joint distinguishes a case of osteoarthritis of the hip from sciatica Acute osteoarthritis can usually be distinguished from acute rheumatic fever by the failure of response to salicylates

**Prognosis** This is unfavourable as regards recovery, but the disease does not progress to a stage of complete crippledom

**Treatment.** A causative septic focus is rarely found Massage

and radiant heat increase the vascularity of the structures around the joint and help to relieve pain. The joint should be rested as much as possible, the patient lying down if the knees or hips are affected for at least an hour daily. No strain should be put on the affected joints, but they should be put through their full range of movement two or three times a day. Exercises which produce discomfort lasting for more than 2 hours are excessive. If the patient is obese, and the knees and hips are involved, dietetic restrictions should be imposed. Vitamin B, administration as for the rheumatoid arthritis (see p. 612) appears beneficial in some cases. Thyroideum gr.  $\frac{1}{2}$  to 1 t.d.s. is of value in cases associated with the climacteric or thyroid deficiency. Gold salts are of no value. Physical treatment such as infra-red rays, diathermy, and paraffin wax baths, may be beneficial by increasing the blood supply. In addition, if only one joint such as the hip is involved, operative measures may be considered, such as moving the joint under an anæsthetic, osteotomy of the femur to correct deformity, removal of foreign bodies from the joints, excision of the bony outgrowths which are preventing movement (cheilectomy), arthroplasty and arthrodesis. Surgical appliances such as Thomas' walking caliper, will take the weight of the body from the pelvis off the hip.

### Rheumatoid Spondylitis

(*Atrophic Spondylitis. Spondylitis Ankylopoietica. Von Bechterew's Disease. Strumpell-Marie Disease. Spondylose Rhizomélique*)

**Definition.** A rheumatoid type of arthritis affecting the spine.

**Etiology.** This is uncertain, but it is usually considered to be the same as for rheumatoid arthritis. *Predisposing causes:* 1. Age: 20 to 30 years. 2. Sex: Males predominate.

**Pathology.** There is synovitis of the posterior intervertebral joints, with osteoporosis of the vertebral bodies. The sacro-iliac and costo-vertebral joints are often affected. Calcification occurs in the ligaments of the spine, and in the lateral borders of the intervertebral discs. The spine thus becomes rigid (bamboo spine).

**Clinical Findings.** The onset is usually insidious with pain and stiffness in the back, especially on waking in the morning. Root pains may occur in the arms or legs, or girdle pains around the chest or abdomen. In a few cases there is an acute onset with fever. *On Examination:* There is rigidity of the back (poker back), the spine may be curved with kyphosis in the upper thoracic region. In the early stages there is considerable spasm of the erector spinæ muscles. Flaccid paresis of the legs may occur from root pressure, with paræsthesia, or cord compression may give rise to spastic paralyses. There may be acute pain on percussion over the lateral spinous processes. Later the patient may be unable to stand upright, to turn without moving the whole body, or to take a deep breath. The sedimentation rate of the red cells is increased. X-ray examination shows decalcification of the bodies of the vertebrae, the joint spaces are narrowed or blurred and the spinal ligaments calcified.

**Differential Diagnosis** The diagnosis is established by the X ray examination of the spine. If there are no bony changes the stiffness is usually due to fibrositis of which chronic gonorrhoea may be the cause. A spinal tumour or syphilitic meningitis may cause similar symptoms, but there are no bony changes.

**Course and Complications** The course is usually slowly progressive. Respiratory complications such as bronchitis render the outlook more unfavourable. Rheumatoid arthritis may occur in other parts of the body.

**Treatment** The general treatment resembles that advised for rheumatoid arthritis. A spinal brace should be worn to relieve the strain on the back. X ray treatment is particularly valuable in the early stages for relief of pain, increasing mobility and preventing deformity.

### Osteoarthritic Spondylitis (*Hypertrophic Spondylitis*)

**Pathology** Degenerative changes occur in the spine similar to those described under osteoarthritis (see p. 613).

**Clinical Findings** Although osteoarthritic changes are present in the spine in the majority of people over the age of 50 they rarely give rise to symptoms. The chief symptoms are root pains, some rigidity of the spine and muscular atrophy. When the cervical spine is involved there may be pain in the shoulders, neck and arms with limited spinal movement.

**Treatment** This includes rest to the spine, heat, massage and in some cases a spinal support.

### Still's Disease

**Definition** A variety of rheumatoid arthritis occurring in children, and characterised by swelling of joints, enlargement of the spleen and lymph glands with pyrexia. A similar condition may affect adults, when it is known as Felty's syndrome.

**Clinical Findings** The disease usually begins before the second dentition. The parents notice swelling in the joints, usually in the hands, knees or wrists, and the child complains of pain and stiffness in them.

**On Examination** The joints show periarticular swelling, the finger joints may be spindle shaped and movement of the joints is painful and limited. The wrists, knees and cervical vertebrae may also be affected. Enlarged glands may be felt generally distributed and the spleen may be palpable. The muscles near the affected joints atrophy, the child is pale, sweats and runs an irregular temperature of about 100° F.

**Course and Complications** In the majority of cases the disease pursues an irregular course with remissions and exacerbations and proceeds to a fatal issue from some intercurrent disease. Internal adherent pericardium may occur.

**Prognosis** This is grave, but recovery has been recorded.

**Treatment.** The patient must be kept in bed during the active stages of the disease. If the tonsils are infected they should be removed. The general condition of the child should be improved by the administration of cod-liver oil, m. 30, t.d.s., and of tonics such as liq. arsenicalis, m. 1 to 2 t.d.s. p.c. Gold salts should not be given. Some encouraging results have been obtained by autohæmotherapy, injecting 5 mls of the patient's blood, removed from a vein, into the gluteal muscles every week. Sulphathiazole (M. & B. 760) has also been recommended in doses proportionate to the age of the patient.

### Specific Infective or Toxic Arthritis

The inflammation of joints is here due to infection with known organisms, or occurs as a complication of infective diseases. This group includes pneumococcal, tuberculous, gonococcal, staphylococcal and streptococcal arthritis, acute rheumatic fever and arthritis associated with syphilis, dysentery (bacillary), Malta fever, dengue, typhoid fever, cerebro-spinal fever, scarlet fever, measles and mumps.

Other varieties of arthritis are as follows: Metabolic, as in gout (see p. 638). Hæmorrhagic, as in purpura and hæmophilia (see p. 521). Nervous, as in Charcot's joints (see p. 390), or in syringomyelia (see p. 415). Anaphylactic, as in serum disease (see p. 541). Associated with deficiency diseases, as rickets or scurvy (see pp. 619, 621). Traumatic and intermittent hydrarthrosis. Here periodical swelling occurs in joints, especially the knees and wrists. There is considerable weakness, but little pain. The swelling usually subsides in 2 to 3 weeks. It is possibly allied to angio-neurotic œdema.

Treatment consists in rest and firm bandages.

## CHAPTER X

### DISORDERS OF METABOLISM

#### DEFICIENCY DISEASES

**Introductory** Certain disorders are believed to be due to a deficiency or absence of a vitamin from the food. They are Xerophthalmia, night blindness, rickets, habitual and threatened abortion, certain myopathies, beri beri, rosacea keratitis, pellagra, scurvy, bleeding associated with jaundice, and neonatal hemorrhage. Vitamins, or accessory food factors, are substances present in food which are essential for growth or health. Only minute quantities are necessary. They fall into two groups according to their solubility in fats or water. The following are described. *Vitamin A* (Growth Vitamin, anti-infective, anti-xerophthalmic) This is derived from green leaves, where it is probably synthesised with the aid of light. It can also be formed apart from the action of light. It is present in animal fats, in milk, butter, cod liver and halibut liver oil, etc. as the result of food eaten by the animal. It is found in traces only in vegetable oils. Carotene ( $C_{40}H_{56}$ ) which is present in vegetables is its precursor, or the pro-vitamin. Some authorities consider that carotene is converted into vitamin A in the liver and stored there. The vitamin A content of milk is lowest in January and highest in June. Vitamin A ( $C_{44}H_{56}O$ ) is destroyed by sunlight, or by heating in air. It gives a blue colour with arsenic or antimony trichloride. The "blue unit" or Carr Price unit is based on this colour change. The international unit is 0.6  $\gamma$  of  $\beta$  carotene. An adult requires 2,000 i.u., and a child 3,000 i.u. daily. A teaspoonful of cod liver oil contains 6,400 i.u. and a drop of halibut liver oil 3,200 i.u. Absence of this vitamin from the food of young animals arrests their growth, the effect produced in adult animals and possibly in men by deficiency of this vitamin in the food is to increase the liability to infections especially xerophthalmia and puerperal sepsis. Night blindness also results from vitamin A deficiency. Excess of the vitamin is harmless.

*Vitamin D* (Anti-rachitic Vitamin) This is present in animal fats such as cod liver and halibut liver oil, but butter is poor in it. It is present in vegetable oils. It is not destroyed by heat and probably not by sunlight, and it does not give a blue colour with arsenic chloride. It can be synthesised from ergosterol, an impurity of cholesterol, by ultra violet rays. Ergosterol is regarded as the pro-vitamin D. Calciferol (vitamin D<sub>2</sub>) prepared from ergosterol, possesses a very high anti-rachitic activity, 1  $\mu$ , 40,000 i.u. per mg., and is closely related to the naturally occurring vitamin D. The international unit of vitamin D is equivalent to 1/10,000 mg. of irradiated ergosterol. An adult requires 150 i.u., and an infant 1,500 i.u. daily. A teaspoonful of cod liver oil contains 480 i.u., and a drop of halibut liver oil 48 i.u. Vitamin D is



**Treatment.** The patient must be kept in bed during the active stages of the disease. If the tonsils are infected they should be removed. The general condition of the child should be improved by the administration of cod-liver oil, m. 30, t.d.s., and of tonics such as liq. arsenicalis, m. 1 to 2 t.d.s. p.c. Gold salts should not be given. Some encouraging results have been obtained by autohæmotherapy, injecting 5 mls of the patient's blood, removed from a vein, into the gluteal muscles every week. Sulphathiazole (M. & B. 760) has also been recommended in doses proportionate to the age of the patient.

### Specific Infective or Toxic Arthritis

The inflammation of joints is here due to infection with known organisms, or occurs as a complication of infective diseases. This group includes pneumococcal, tuberculous, gonococcal, staphylococcal and streptococcal arthritis, acute rheumatic fever and arthritis associated with syphilis, dysentery (bacillary), Malta fever, dengue, typhoid fever, cerebro-spinal fever, scarlet fever, measles and mumps.

Other varieties of arthritis are as follows: Metabolic, as in gout. (see p. 638). Hæmorrhagic, as in purpura and hæmophilia (see p. 521). Nervous, as in Charcot's joints (see p. 390), or in syringomyelia (see p. 415). Anaphylactic, as in serum disease (see p. 541). Associated with deficiency diseases, as rickets or scurvy (see pp. 619, 621). Traumatic and intermittent hydrarthrosis. Here periodical swelling occurs in joints, especially the knees and wrists. There is considerable weakness, but little pain. The swelling usually subsides in 2 to 3 weeks. It is possibly allied to angio-neurotic œdema.

Treatment consists in rest and firm bandages.

## CHAPTER X

### DISORDERS OF METABOLISM

#### DEFICIENCY DISEASES

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believed to be synthesised in man by the action of sunlight on ergosterol present in the subcutaneous fat. It is also probably synthesised in fish apart from the action of light and is not derived from their food. Absence of this vitamin from the food of puppies is followed by rickets. In human beings deficiency of this vitamin may cause rickets or dental caries, and overdosage results in irritability, anorexia, diarrhoea, excess of calcium in the bones and deposition of calcium in the kidneys.

**Vitamin E.** (Fertility or anti-sterility Vitamin.) This fat-soluble vitamin is present in olive oil, wheat embryo and green leaves. Wheat-germ oil is its most potent source. Chemically it is a mixture of  $\alpha$ - and  $\beta$ -tocopherol. Clinically, good results have been obtained in some cases of habitual and threatened abortion by the administration of wheat-germ oil extract, m. 3 capsules, 1 daily for 3 to 6 months before the onset of labour. Synthetic  $\alpha$ -tocopherol has also been used in the treatment of certain myopathies. It is essential for reproduction in rats.

**The Vitamin B Complex.** This vitamin has been divided into at least four factors. It occurs in seeds, eggs, the germ and bran of cereals including the husk of rice, and in yeast, meat, fish, milk, etc. Its presence in bread varies with the amount of wheat-germ used.

**Vitamin B<sub>1</sub>.** This water-soluble vitamin has been isolated in pure form and also synthesised. The hydrochloride is known as aneurine or thiamine. An adult requires about 700 i.u. daily, and 3 mg. of synthetic vitamin B<sub>1</sub> hydrochloride are equivalent to 1,000 i.u. Deficiency leads to beri-beri and possibly to certain types of peripheral neuritis in man and to polyneuritis in pigeons. It is believed that minor degrees of deficiency will cause in animals and man anorexia, constipation, digestive disturbances and liability to intestinal intoxication. Pink disease in children may be due to lack of vitamin B<sub>1</sub>.

**Vitamin B<sub>2</sub>.** This contains riboflavin (lactoflavin), and nicotinic acid. **Riboflavin or Lactoflavin.** Lack of this vitamin may be responsible for the glossitis and angular stomatitis met with in pellagra. Rosacea keratitis also responds to its administration.

**Nicotinic acid** (Vitamin P.P. or pellagra-preventing). In addition to its use in the prevention and treatment of pellagra (see p. 625) it is possible that Vincent's angina is a pre-pellagrous condition (see p. 2).

**Vitamin B<sub>6</sub>** (Adermin). This is the anti-dermatitis factor in rats. Some cases of pseudo-hypertrophic muscular dystrophy in man have been improved by its administration.

**Vitamin C** (Anti-scorbutic Vitamin). The following are particularly good sources of vitamin C:—Hips, haws, blackcurrants and Brussels sprouts. Sprouts should be cooked by cutting them up and dropping them into boiling water, taking care the water does not go "off the boil." The vitamin is not then likely to be destroyed by oxidases during cooking. It is now believed that ascorbic acid is the vitamin. It is destroyed by heat in the presence of air if the reaction is alkaline. It can be prepared in bulk from certain peppers of the genus capsicum. It is generally absent from tinned fruit, but it is present in fruit canned anaerobically, as heat does not destroy it in the absence of oxygen.

Adults require 50 to 100 mg of ascorbic acid (1,000 to 2,000 i.u.), and infants 15 to 50 mg of ascorbic acid daily. Its deficiency causes scurvy in infants and in adults and possibly faulty enamel formation in teeth.

**Vitamin K** This is widely distributed amongst plants and a synthetic substance, 2 methyl 1,4 naphthoquinone, is available for clinical use as Kapilon and Prokayvit. Klotogen is an oily concentrate of the naturally occurring vitamin. It is valuable in checking hæmorrhage associated with jaundice and neonatal hæmorrhage (see pp. 86, 523). There is deficient absorption of vitamin K when bile is absent from the intestine.

**Vitamin P** This is present in Hungarian red pepper, lemon juice and orange peel. It is available for clinical use as Hesperidin in the treatment of certain cases of petechial hæmorrhage.

### Rickets

**Definition** A general metabolic disorder of infants, with characteristic changes in the bones, especially near the epiphyses.

**Etiology** Rickets may be caused by 1 Errors of diet, especially deficiency of vitamin D (see p. 617) in the milk. Excess of cereals produces an antagonistic effect on vitamin D. 2 Absence of sunlight. The ultra violet rays are antirachitic and perhaps effect a synthesis of the vitamin D (see p. 618). They do not pass through ordinary glass. 3 Defective hygiene and confinement. *Predisposing causes* 1 Age 6 to 18 months. 2 Season Winter and spring. 3 Climate and locality. In the temperate zones, and in large cities. 4 Absence of breast feeding. Rickets, however, often does develop in a breast fed baby. 5 Prolonged breast feeding. After 1 year. 6 Rapid growth.

**Pathology** *The Bones* The calcium phosphate content is low, about 21% instead of the normal 63% and, owing to lack of calcium, the bones bend. Changes are seen in the long bones, near the epiphyses, the end of the bone is enlarged and the epiphyseal line is widened beyond the normal 2 mm. and uneven, owing to irregular and excessive proliferation of the cartilage cells. Normal ossification does not ensue, and the marrow is unduly vascular. The deficiency of calcium and phosphorus in the bone is probably due to failure of absorption from the intestine. The blood phosphorus and calcium are usually low in active rickets, such as Ca. 6 and P 2 mg per 100 c.c. (normal figures Ca 10 and P 5 mg per 100 c.c.). It is believed that calcium phosphate is normally deposited in growing bone owing to the action of an enzyme, phosphatase, formed in bone. Phosphatase also occurs in the intestinal mucous membrane, in the kidneys and in the blood. The normal plasma phosphatase figure is 3 to 13 units (King and Armstrong method). In rickets the plasma phosphatase is increased during the active stages of the disease. The liver may be enlarged and fatty and the spleen fibrous. As the condition improves the calcium and phosphorus contents of the blood rise and the phosphatase content falls.

**Clinical Findings** The patient is usually an infant, aged about a year, a history of improper feeding may or may not be obtained, the

symptoms are generally noted in the winter or spring; they include irritability, restlessness and sweating on the head at night; the child may also suffer from diarrhoea, bronchitis, convulsions, tetany or laryngismus stridulus; in many cases he is not brought for treatment until the disease is more advanced and advice is sought on account of delay in walking, in speaking or in dentition, or for curving of the limbs.

*On Examination:* The child is usually pale but well covered. The earliest sign of rickets is craniotabes, which can be detected in a premature baby at the age of 1 to 3 months. Softened areas of bone are felt, especially behind the ears. A month or so later enlargement of the lower end of the radius, ulna and femur and of the upper end of the tibia may be noted, with beading of the costo-chondral junction. A transverse depression running round the front of the lower part of the chest, called *Harrison's sulcus*, is seen at the level of the diaphragmatic attachment.

Prominence of the sternum may cause a pigeon chest appearance. Bossing of the skull is due to prominence of the frontal and parietal eminences (hot-cross bun appearance); there is delay in closure of the anterior fontanelle (normally closed in 18 months). The legs, arms or spine may be curved, and the pelvis flat; green stick fractures may occur in the extremities. The muscles are weak and the ligaments often lax. The liver and spleen are palpable, due either to displacement secondary to chest deformity or to enlargement. The abdomen is distended. Rhonchi may be heard in the lungs. The blood usually shows anæmia, and the phosphorus and calcium are low and the phosphatase is raised as mentioned above. X-ray examination: The earliest bone changes are the disappearance of the sharp margin of the metaphysis at the lower end of the radius, ulna or femur, later the diaphyseal end becomes cupped and the epiphyseal line is widened.

*Varieties:* Adolescent Rickets (*Rachitis Tarda*); rickety manifestations appear at puberty, affecting only the long bones. The disease is associated with conditions of great privation. Adult rickets is exemplified by osteomalacia.

*Differential Diagnosis.* Congenital syphilis may be suggested by the appearance of the skull, and the curving of the bones; the Wassermann reaction of the blood differentiates. The enlarged head must be distinguished from that due to hydrocephalus, the curved spine from that produced by caries, and the muscular weakness from that caused by poliomyelitis or scurvy.

*Course and Complications.* The course depends largely on the treatment; if untreated there is a tendency to cure after the second year of life, but bony deformities persist. Complications include: Bronchitis, bronchopneumonia, laryngismus stridulus and possibly tetany. Permanent deformities may result if treatment is delayed or inadequate, with dwarfism and difficulty in childbirth.

*Prognosis.* This is very good, provided the rickets is diagnosed before an advanced stage is reached and that it is treated efficiently. Death may occur from convulsions or from laryngismus stridulus.

Rickets increases the gravity of measles and bronchopneumonia in infants

**Treatment** *Prophylactic* Expectant mothers should have a correctly balanced dietary. Every baby at the age of three weeks should be given vitamin D, 180 i.u. daily, such as cod liver oil m 20 t.d.s. or halibut liver oil 4 drops t.d.s. Premature babies require double the amount. The dose should be gradually increased to 1,500 i.u. daily, and thus maintained for several years. Raw yolk of egg is also a good preventive. One half may be given daily for the first 2 months, and the whole yolk daily after this. The baby should be out of doors every day, and the arms and legs exposed to the sun. In winter, if the baby cannot take cod liver oil he should have general ultra violet light radiation every other day. One and a half pints of milk should be given daily during the second year of life.

*Curative* The child should be taken off his feet during the acute stage and treated with 5,000 i.u. of vitamin D daily (= m 625 of cod liver oil or 104 drops of halibut liver oil). Concentrated preparations of irradiated ergosterol are available such as Haliverol, and calciferol. Haliverol (halibut liver oil with added vitamin D) contains 250 times as much vitamin D as does cod liver oil. Calciferol is put up as liq. calciferol (B.P. Add.) m 5 = 1,000 i.u. Ultra violet light, or irradiated milk 12 to 18 oz. daily with an equal amount of unirradiated milk for a child of 2 to 5 is helpful in some cases. Overdosage (hyper-vitaminosis) must be avoided, as the bones may become prematurely ossified and calcium be deposited in the kidneys. If there is marked deformity of the limbs splints may be required, this also keeps the child off his feet. The limbs should be massaged. X-rays show evidence of healing first at the end of the ulna, radius or femur, as a new calcification zone between the epiphysis and diaphysis. Diet. Excess of starchy foods must be avoided. The diet should consist of milk 1½ pints, rusks made from wholemeal bread, yolk of an egg, orange juice and a little porridge, gravy, greens and steamed fish.

## Infantile Scurvy

### (Barlow's Disease)

**Definition** A disease characterised by hæmorrhages into the skin, mucous membranes and under the periosteum without characteristic blood changes.

**Etiology** Infantile scurvy is due to a deficiency of vitamin C in the diet (see p. 618). It is a disease of infancy, manifesting itself between the ages of 8 and 12 months. It develops in infants fed on boiled pasteurised, dried or condensed milk who have not been supplied with the vitamin as should be done by giving fruit juice.

**Pathology** There is increased capillary permeability. At autopsy hæmorrhages may be found under the periosteum especially of the long bones, the epiphyses may be separated or the bones fractured. There is inhibition of the growth of osteoblastic bone, and the cartilage

is less vascular than normal. There may also be infarcts in the lungs and hæmorrhages into the intestines or kidneys.

**Incubation Period.** The disease manifests itself after deprivation of the vitamin for 6 to 8 months.

**Clinical Findings.** The onset is usually gradual, and the baby may be brought to a doctor on account of screaming when the limbs are touched, or because he does not move an arm or leg, or for pallor, bruising on the face near the orbit, prominence of an eye, or bleeding from the gums, bowel or urinary tract. A history of improper feeding is usually obtainable.

**On Examination :** The infant may be pale, wasted and fretful, crying when touched. Bruising may be seen on the face or body, or petechiæ on the palate. Hæmorrhages are only seen from the gums in the neighbourhood of erupted teeth. An arm or leg may appear paralysed, the child not moving the limb owing to pain. A tender swelling may be felt deep to the muscles, often towards the lower end of the femur. There may be œdema of the face or limbs, and the urine may contain blood or a trace of albumin. There may be proptosis of an eye, due to retro-orbital hæmorrhage. The temperature is usually a little raised, about 100° F. and the pulse frequent. The blood : This shows an anemia, but the platelet count, bleeding time, and coagulability are normal. The plasma phosphatase (see p. 619) is low. X-ray examination may reveal a subperiosteal swelling, separated epiphysis or fracture. Frænkel's "white line" is a dense transverse shadow at the junction of the epiphysis and diaphysis.

**Differential Diagnosis.** The following conditions should be considered :—Purpura or leukæmia : The gums are not affected in purpura and there is no dietetic error. In leukæmia the blood count is diagnostic. Osteo-myelitis and poliomyelitis : The presence of hæmorrhages are characteristic of scurvy ; the X-ray appearances also help to differentiate osteo-myelitis ; there is no real paralysis present, and the deep reflexes are unaltered. Acute rheumatism does not occur below the age of 2 years. Syphilitic epiphysitis is usually seen under the age of 3 months. Trauma may be suggested by the bruising, or a retro-orbital growth if there is proptosis.

**Course and Complications.** Adequate treatment rapidly checks the course of the disease. Complications include :—Bronchopneumonia, otitis media, furunculosis, nasal diphtheria and diarrhœa. Rickets may also be present (scurvy rickets).

**Prognosis.** The infant should be cured in a week with proper treatment.

**Treatment. Prophylactic.** Vitamin C must be supplied from birth, in the form of orange, blackcurrant, or rose hip juice, sweetened and diluted with water. The protective daily dose for an infant is ascorbic acid mg. 15 to 50. 50 mg. of ascorbic acid is contained in 3 oz. of fresh orange juice, 1 oz. of blackcurrant juice, or  $\frac{1}{2}$  oz. of rose hip juice.

**Curative.** Orange, blackcurrant or rose hip juice should be given daily as above, and in addition ascorbic acid tab. mg. 50, should be

given t i d While the limb is painful the baby should be disturbed as little as possible and the limb supported on a splint

### Adult Scurvy

**Etiology** James Lind, an officer of the British Navy, showed in 1753 that scurvy is not an occupational nor an infective disorder, but is due to the lack of certain foodstuffs He introduced lemon juice into the seamen's ration Scurvy ravaged Paris during the siege in 1870 and was met with in Russia, Rumania and Mesopotamia during the 1914-18 war

The juice of sweet limes or lemons from the Mediterranean is preventive, but the sour limes from the West Indies have not this property Seeds such as barley, peas, beans, or lentils acquire antiscorbutic powers if soaked in water for 24 hours, and kept moist for 3 days They then germinate, and the sprouts are antiscorbutic Soda, used in cooking green vegetables to preserve their colour, destroys their antiscorbutic properties

**Clinical Findings** The sufferer complains of general weakness, shortness of breath and vague limb pains Hæmorrhages are then noted from the gums or nose, and there may be night blindness

**On Examination** The patient is sallow, the gums may form fungating masses obscuring the teeth which become loose, the breath is foul tender lumps due to hæmorrhages may be felt in the skeletal muscles, and hæmorrhages are seen in the skin Ascorbic acid is usually absent from the urine

**Treatment** This consists in supplying antiscorbutic substances, as described above, or by the subcutaneous injection of ascorbic acid mg 100 to 300 t i d Hydrogen peroxide should be applied to the gums

### Beri-beri

#### (Polyneuritis Endemica)

**Definition** A deficiency disease characterised by polyneuritis, cardiac symptoms and œdema

**Etiology** Beri beri is due to deficiency of vitamin B<sub>1</sub> (see p 618) in the diet This deficiency may result in the production of a metabolic toxin The disease occurs in countries where the chief article of food is polished rice deprived of the pericarp which contains the vitamin It is thus endemic in the Malay States, Japan and the East Indies It may also occur on sailing ships if the diet is deficient in vitamin B<sub>1</sub>

**Pathology** At autopsy the heart weighs more than normal, there is enlargement chiefly of the right ventricle Serous effusions occur in the pericardium and pleura The liver and spleen are congested and the lungs œdematous The mucous membrane of the stomach and duodenum may be congested The nervous system There may be degeneration of anterior horn cells in the cord and of posterior ganglion cells In the peripheral nerves the myelin sheath degenerates and the axis cylinder may rupture, the sympathetic fibres may also degenerate,



and vagus and phrenic neuritis may occur. The heart failure is possibly due to water retention in the cardiac muscle, rather than to vagal neuritis.

**Incubation Period.** Symptoms usually appear after deprivation of vitamin B<sub>1</sub> for 3 months.

**Clinical Findings.** There are two main types of the disease, "dry" and "wet." The patient is usually a young adult, but infants of mothers suffering from beri-beri may show symptoms of the disease. The patient complains of weakness, numbness or tingling in the legs, dyspnoea on exertion, palpitations, tender spots in the calves and swelling of the legs.

**On Examination:** In the "dry" type signs of peripheral neuritis are found, such as weakness and wasting of the leg muscles, areas of cutaneous anaesthesia or hyperaesthesia, depression or loss of the deep reflexes, and the reaction of degeneration is obtained. Localised tender swellings may be present in the calf (probably due to water retention in the muscles). The skin is dry. In the "wet" variety, oedema is present in the legs and free fluid may be found in the abdomen, pleurae or pericardium. The heart is enlarged chiefly to the right, the apex beat becomes diffuse and fluttering, the rhythm remains regular, and the pulse is soft and unduly frequent. The electrocardiogram shows no abnormality beyond right axis deviation. The cervical veins are engorged and the liver may enlarge and pulsate. Oedema of the lungs is a terminal event. In addition neuritis may be present. The temperature usually remains normal. An acute variety is described in which the patient dies in a day or so from heart failure. Rudimentary forms also occur, with recurrent pain or weakness in the legs.

**Differential Diagnosis.** The occurrence of oedema and peripheral neuritis with heart symptoms, in a locality in which beri-beri is endemic, is diagnostic. Peripheral neuritis due to other causes must be excluded in the "dry" variety and a careful enquiry made into the diet. Vitamin B<sub>1</sub> deficiency in the diet can be detected by estimation of its excretion in the urine.

**Course and Complications.** The course is usually progressive unless the dietetic error is corrected.

**Prognosis.** Death may occur suddenly from heart failure. Recovery is the rule with adequate treatment.

**Treatment. Prophylactic.** An ordinary mixed diet is all that is requisite; in rice-eating countries, some of the husk of the rice should be left after milling. Yeast or Marmite contains the vitamin and can be eaten when fresh food is scarce, or white flour used in the making of bread may be fortified with synthetic vitamin B<sub>1</sub>.

**Curative.** The vitamin B<sub>1</sub> can be supplied in the form of yeast, Marmite, rice bran, cereal bran, wholemeal bread and milk. As there may be faulty absorption, 5 to 50 mg. of vitamin B<sub>1</sub> may be injected intramuscularly daily for twelve doses in the form of Benerva (aneurin. hydrochlor. B.P.Add.). During the acute stages the patient must be kept in bed; if there is venous engorgement relief is obtained by bleeding. Massage should be given to the extremities.

## Pellagra

**Definition** A disease characterised by dermatitis, gastro intestinal and nervous disturbances

**Etiology.** Pellagra is believed to be due to deficiency of vitamin B<sub>3</sub> (nicotinic acid and riboflavin), vitamin B<sub>6</sub> (adernin) and possibly vitamin B<sub>1</sub> (see p 618) The disease is associated with diets rich in maize, but poor in meat and milk It is possible that a toxin present in maize is also a causative factor *Predisposing causes* 1 Age 20 to 40 years 2 Sex Slightly more common in females 3 Season Chiefly in spring and early autumn 4 Locality Especially Italy, Rumania, America Egypt and India 5 Debilitating conditions such as poverty, recurrent malaria, syphilis, tuberculosis and alcoholism

**Pathology** At autopsy the body is wasted and dermatitis may be seen on exposed areas of skin There is atrophy of the muscle and mucous membrane of the stomach and intestines, and ulcers may be found in the colon The heart is small The liver may be fatty or cirrhotic There is degeneration of the posterior and lateral columns of the cord, but no peripheral neuritis Meningeal thickening may be present in the brain or cord

**Clinical Findings** The patient is usually an adult who notices in the spring loss of appetite, nausea, vomiting or diarrhoea He may also complain of soreness of the mouth or tongue, and excessive salivation Later, redness and itching of exposed areas of the skin may be noted The patient may also complain of numbness or cramps in the legs, and of headache, giddiness or insomnia

**On Examination** Affected areas of skin may be seen on the backs of the hands and forearms, on the forehead on a butterfly area of the face and on the dorsum of the feet They are symmetrical, resembling sunburn, with definite edges They darken, and blebs may form, during the summer the colour fades and the skin desquamates, and after several years the affected skin becomes thin Recurrence may occur in the autumn or not until the next spring The tongue is often red and glazed, and angular stomatitis is seen, a fractional test meal may reveal a benign type of achlorhydria, blood or mucus may be seen in the motions A spastic or flaccid paresis may be found in the legs the former being associated with achlorhydria The cerebro spinal fluid is normal but the blood often shows an anæmia The temperature is usually normal An acute variety of the disease may occur but the cases described as pellagra sine pellagra are of doubtful etiology

**Differential Diagnosis** The skin lesions are diagnostic and differentiate pellagra from such diseases as sprue or spastic paralysis due to other causes

**Course and Complications** There is always a tendency to seasonal recurrences of increasing severity, wasting and mental deterioration in these cases gradually ensue, with permanent paralysis.

**Prognosis** Apart from the acute cases which survive for only a few weeks, patients often live for 20 years or longer after the diagnosis

is made. Many cases die in the asylum with delusions or dementia, if adequate treatment has not been instituted early.

**Treatment. Prophylactic.** A well-balanced diet is essential, containing lean meat, fruit, vegetables, eggs, milk and butter.

**Curative.** During the acute stages the patient should be put to bed, and nicotinic acid, mg. 100, given 5 times a day in addition to a mixed dietary which includes fresh milk, meat, liver, eggs, fruit and no maize. If there is a sore tongue and angular stomatitis, riboflavin, mg. 1, should be given t.i.d. For diarrhoea and achlorhydria dilute hydrochloric acid should be given in m. 30 to 60 doses diluted with 4 oz. of water and a little syrup of orange three times a day after meals. Injections of liver extract should also be given, or stomach extract taken by mouth (see p. 490). Peripheral neuritis should be treated by daily intramuscular injections of mg. 50 to 100 of Benerva (aneurin. hydrochlor. B.P.Add.). The skin should be protected from the sun, and for the moist lesions pot. permang. solution, 1 in 5,000, should be applied. Good results have also been obtained by intravenous injections of sod. thiosulphate, 10 mls of a 10% solution being given daily for 20 to 60 days.

### Famine or War Œdema

A condition of œdema of the legs may be met with amongst prisoners of war due to a lack of protein in the diet. It also occurs in civilian life when the chief article of diet is alcohol. The plasma proteins are low and fluid passes by osmosis into the subcutaneous tissues.

**Treatment.** Blood transfusions of 300 to 500 mls should be given twice a week and an adequate intake of protein ensured.

### Glycosuria

Glucose is found in the urine as the result of alterations in the secretion of the ductless glands, deficiency in the body storage mechanism, or lowering of the renal threshold for sugar.

**Etiology.** 1. *Affections of the Ductless Glands.* Pancreatic lesions causing deficiency in insulin as in diabetes mellitus or hæmochromatosis. Hyperthyroidism (see p. 649). Hyperpituitarism (see p. 668). Hyperadrenia (see p. 663).

2. *Cerebral Lesions,* such as tumours, hæmorrhage or meningitis, and experimental piqure of the floor of the fourth ventricle. The glycosuria probably results from reflex stimulation of the adrenals and liver through the splanchnic nerves.

3. *Storage Deficiency.* The liver may not be able to store completely the ingested glucose as glycogen, with resultant hepatic glycosuria. A "lag" curve (see Fig. 61) indicates a defect in the storage mechanism, or an abnormally rapid absorption (see p. 629). In health it is very doubtful whether alimentary glycosuria can occur, i.e., glycosuria after a meal rich in carbohydrate.

4. *A Low Renal Threshold.* If the renal threshold for glucose is below the normal of 180 mg. glucose per 100 c.c. blood, renal glycosuria (see Fig. 61) will ensue.

In all cases in which glucose is found in the urine, it is wise to determine the glucose tolerance (see p 629) as this will indicate the severity of the condition

### Diabetes Mellitus

**Definition** A disease characterised by glycosuria, hyperglycæmia and a disturbance of carbohydrate, fat and protein metabolism

**Etiology** Diabetes mellitus is due to a deficiency of insulin, the internal secretion of the pancreas *Predisposing causes* 1 Age Diabetes occurs at all ages, but less commonly at the extremes of life, 80% of all cases are over the age of 40 years 2 Sex Females predominate 3 Heredity There is a tendency to a familial incidence, but the disease is not congenital 4 Race Jews are prone to diabetes 5 Habits Overeating and lack of exercise Eighty per cent of diabetic patients are obese when the disease is first discovered, but this does not apply to children 6 Nervous shock.

**Pathology** Insulin is derived from the islets of Langerhans In diabetes the blood sugar is above the normal range of 80 to 120 mg per 100 c c The sugar tolerance is diminished, as shown by the curve of blood sugar readings obtained after giving 50 G of dextrose by mouth to the fasting patient, and estimating the blood sugar every half hour Glucose is present in the urine, and acetone and diacetic acid may also be found there *The pancreas* It is usually thought that diabetes mellitus results from damage to the  $\beta$  cells of the islets of Langerhans The pathological changes, however of fibrosis hyalinization and hydropic degeneration are only found to a marked degree in about 21% of cases, and in about 23% the pancreas appears normal *The pituitary* The well known Houssay experiment showed that if a depancreatized dog is hypophysectomized the diabetes disappears and hypoglycæmia may ensue If an injection of an extract of the anterior lobe of the pituitary is now made into such a Houssay dog, death will result from glycosuria and ketonuria This is the basis of the belief that diabetogenic and ketogenic hormones are formed in the anterior lobe, and it suggests that in man the pituitary may play a part in the production of diabetes It was later shown that repeated injections of an extract of the anterior lobe will produce permanent diabetes in the dog, which is not associated with ketonuria The pancreas in such a case shows an absence of islets or a reduction in the number of the  $\beta$  cells (See also pituitary glycosuria on p 669) *The liver* Usually no changes are found The metabolism of carbohydrate, protein and fat is disturbed The carbohydrate which is absorbed from the intestines in the form of monosaccharides (glucose, levulose and galactose) is not stored efficiently in the liver as glycogen, and is not used adequately by the muscles but is excreted in the urine as glucose Protein The metabolism is disturbed as the sugar portion of glyco protein is excreted and not metabolised Fat metabolism Without efficient deposition of glycogen in the liver acetone bodies are formed in excess in the liver Acidosis or ketosis results, with diminution in the alkali reserve in the

body and blood. Some authorities believe that coma due to ketosis does not occur apart from sepsis.

**Clinical Findings.** The patient is usually an adult who seeks medical advice for such symptoms as general weakness, wasting, pruritus vulvæ, balanitis, thirst, polyuria, pains in the legs, boils and carbuncles, gangrene of a toe, or constipation. In some instances failure of sight or impotence is first complained of, or the patient may be seen for the first time in a state of coma.

**On Examination:** The patient may be well nourished (*diabète gras*)

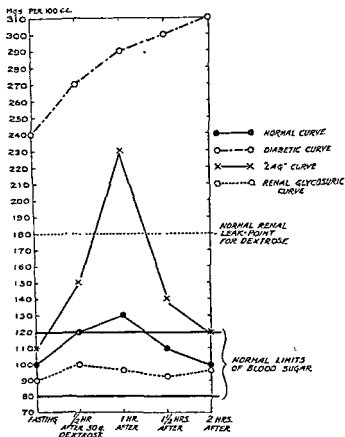


FIG. 61. CHART SHOWING SUGAR TOLERANCE CURVES.

or thin (*diabète maigre*). The tongue may be red and dry and the complexion high coloured. In the degenerative type of case the patient is usually over middle age and signs of cardio-vascular degeneration are present. The urine: The specific gravity is usually over 1,020, and the reaction is acid. A copper-reducing substance is present, as shown by Fehling's or Benedict's test. Special laboratory tests demonstrate that the substance is glucose. Acidosis may or may not be present, as shown by Gerhardt's ferric chloride test and the more delicate sodium nitroprusside test of Rothera. The blood: Sugar is present above the normal

range of 80 to 120 mg per 100 cc in a specimen taken when the patient is not fasting, the amount of blood sugar present is usually proportionate to the severity of the disease. The alkali reserve indicates the presence or absence of acidosis. The normal figure for the  $\text{CO}_2$  capacity of the blood is 53 to 77 cc  $\text{CO}_2$  per 100 cc plasma. With acidosis lower figures are obtained.

**Coma due to Ketosis** At the onset the patient may complain of abdominal pain, constipation, nausea, vomiting or restlessness. The chief clinical features during coma are air hunger (Kussmaul's type with slow and deep respirations), a smell of acetone in the breath, flaccidity of muscles, softness of the eyeballs, loss of deep reflexes, tachycardia of over 120, a subnormal temperature, low blood pressure, a leucocytosis of 15,000 per c mm or over, the urine contains sugar and ketone bodies and the alkali reserve of the blood is low, the blood sugar is high. When there is renal failure the urine may not give the ferric chloride reaction although the breath smells strongly of acetone.

**Differential Diagnosis** The reducing substance in the urine may not be glucose. Thus Fehling's solution is reduced by uric acid, creatinin, glycuronic acid, levulose, lactose, pentose and homogentisic acid. Benedict's solution is not reduced by uric acid or creatinin. Reduction with Benedict's solution therefore indicates a derangement of carbohydrate metabolism with the exception of homogentisic acid and the actual substance present can be identified by special tests. Glycosuria, however, does not necessarily mean diabetes. A blood sugar tolerance test will decide whether diabetes is present and if so, its severity. Typical results are shown in the graphs given (see Fig. 61).

In renal glycosuria, although there is glycosuria, the blood sugar curve shows that the renal threshold is low, sugar appearing in the urine, although the amount in the blood is not above normal. Renal glycosuria may rarely develop later into diabetes, a patient who has this condition should have a sugar tolerance test done once a year, beyond this no treatment is required. A "lag" curve may be obtained. The sugar storage mechanism cannot keep pace with the dextrose absorption. Glycosuria occurs after a meal rich in carbohydrates and the blood sugar curve shows a fall to normal in 2 hours. A "lag" curve may also occur after gastro-enterostomy and in some cases of duodenal ulcer owing to rapid absorption of dextrose from the intestine. This condition may lead on to diabetes.

Renal glycosuria must be distinguished from pentosuria. In both conditions a substance is present in all specimens of the urine which reduces Benedict's solution, and the blood sugar and sugar tolerance curves are normal. Special chemical tests on the urine are required to distinguish between pentose and glucose, the simplest being the fermentation one. Pentose does not ferment yeast, whereas glucose does.

Coma due to ketosis must be differentiated from meningitis or cerebral hemorrhage in which hyperglycemia and glycosuria are often present. If the temperature is normal or above normal the case is probably not one of uncomplicated diabetic coma.

**Course and Complications.** The course depends upon the type of disease present, mild or severe; in children it is usually severe, in elderly people mild. The course can be favourably modified by adequate treatment in the majority of cases. Complications include: Septic lesions such as boils, carbuncles, multiple abscesses in muscular tissues and streptococcal cutaneous or subcutaneous lesions. Pulmonary tuberculosis. Gangrene of the lung. Peripheral neuritis. Myocardial degeneration, with symptoms of heart failure. Gangrene, especially of the toes. Coma, either hyper- or hypoglycæmic, the former being due to acidosis, the latter to overdosage of insulin. Cataract. Retinitis. Chronic nephritis. Korsakow's psychosis (see p. 748).

**Hypoglycæmia.** The patient who is receiving insulin for the treatment of diabetes may suffer from hypoglycæmia. This is particularly liable to occur if a carbohydrate meal is not taken within half an hour of the injection, or if the patient takes an undue amount of exercise. The early symptoms are sweating, flushing or pallor, headache, abdominal discomfort, weakness, tremors, visual disturbances and coldness of the extremities. The patient may vomit and become unconscious with marked convulsions resembling those of acute mania. Hemiplegia sometimes occurs. Children may become pale and then suddenly pass into coma. The hypoglycæmic attack may not occur until about 4 p.m., i.e., after lunch, although no insulin has been given since before breakfast. The treatment is described later (see p. 637).

**Prognosis.** This is good in all mild types of disease; in severe cases the prognosis is usually favourable, provided the patient can be adequately treated and will be conscientious in his after-treatment. Given skilled stabilising treatment the prognosis largely depends upon the patient. Perhaps in no other disease does a good patient reap such a gratifying reward. Any septic focus may predispose to, or excite coma. Diabetes with tuberculosis forms a very fatal combination, and the patient usually requires a large amount of insulin. The insulin required often falls in patients who are meticulous in carrying out the dieting, and in some cases after 2 or 3 years no insulin is required. Death in young people is usually due to coma. Other causes of death are cardiovascular degeneration, sepsis and infection without coma, and intercurrent diseases, especially tuberculosis and cancer.

**Treatment. Stabilising Treatment.** A preliminary blood sugar estimation should be made, to make certain that hyperglycæmia is present. Mild cases usually require no insulin; if complications are present, such as ketosis, neuritis, pulmonary tuberculosis, gangrene of the extremities, etc., insulin will be required and should be administered at once (see below). The patient should be put to bed, the urine tested for glucose and acetone bodies and the blood sugar estimated while he is on a standard diet, of definite caloric value, such as Diet 5 (see p. 613). If the diabetes is not severe, as shown by the absence of complications and ketosis, the patient is then starved for 24 to 48 hours until the urine is sugar free. During this period he is given fluids such as water, lemonade without sugar, Bovril, weak tea or coffee (no sugar and no milk). If a severe case is suddenly starved, coma may be precipitated.

Such a patient should therefore be started on a moderate diet, such as No 3, and insulin given as described later. The graduated diets are then begun and the patient is worked up through them, increasing the diet every second or third day, until he is taking a diet adequate for his needs. This is calculated by determining the number of calories required on the basis of 15 cal's for each 1 lb of "correct" body weight, and adding 10 to 20% more to the figure for the basal requirement diet so obtained, according to the muscular work which will be done when the patient is about again. The "correct" body weight is obtained from a body length (sitting height) and body weight table. Thus a man who should weigh 10 st requires 2 100 cal's, as a basal diet. If the patient cannot reach the necessary diet without sugar appearing in the urine, and the blood sugar rising above the normal limit of 120 mg per 100 c c, insulin will be necessary.

Relation Between Body Length and Body Weight  
(Ainley Walker and Dreyer)

Body Length (inches)	Body Weight (pounds)	
	Males	Females
20	24	24
22	32	32
24	42	43
26	54	55
28	68	70
30	84	89
32	103	108
34	125	131
36	150	157
38	177	186
40	208	219

Constipation must always be corrected medicinally, as its presence appears conducive to coma.

*Administration of Insulin* In private practice it is often impossible for daily blood sugar estimations to be made. They are not necessary, providing care is taken to test specimens of urine frequently, as detailed below. The régime is as follows. 9 a m, specimen of urine collected and insulin injected if sugar is present, 9 30 a m, breakfast, 12 noon, urine collected, 1 p m, lunch, 3 p m, urine collected, 4 30 p m, tea, 6 p m, urine collected, 6 30 p m, dinner, 9 p m, urine collected, 12 midnight, urine collected, 6 a m, urine collected. The specimen passed before an insulin injection is due should always be tested before the insulin is given. Five units of soluble insulin are first injected half an hour before breakfast, and this dose is increased by 2 units at a time until the mid day specimen of urine is sugar free. He is then put on the next diet and the insulin increased, if necessary, until he is again sugar free. In this way the insulin dosage and diets are increased until the selected diet is being taken and the urine remains sugar free in all the specimens. Breakfast contains more carbohydrate than dinner and so



the larger insulin dose is given before breakfast. If the 9 a.m. specimen of urine contains sugar, 5 units of soluble insulin are also given before dinner, and this dose is increased by 2 units at a time until the early morning specimen of urine is sugar-free. It is not advisable to give a single dose of more than 40 units of soluble insulin unless blood sugar estimations are being made, except as an emergency measure in diabetic coma. In children and young adults high carbohydrate diets are usually required (see p. 634). Hagedorn introduced protamine insulinate (Insulin Retard) which is more slowly absorbed, and so reactions are less liable to occur. The insulin effect begins in 1 to 3 hours after injection, and is maximal at 6 to 8 hours. In some cases protamine insulinate produces a remarkable flattening out of the blood sugar curve, in other instances it is disappointing. Protamine zinc insulin is now also available. It contains 40 or 80 units of insulin in each mil. The insulin suspension must be shaken immediately before use. The absorption of the insulin is still further delayed by the zinc, which is present in the proportion of 1 mg. to 500 units of insulin. The insulin effect may begin within 3 to 6 hours after injection, and it lasts for 15 to 60 hours. In order to prevent the blood sugar rising after a meal containing 40 G. or more of carbohydrate it may be necessary to inject a small dose of soluble insulin simultaneously with the zinc suspension insulin. It is generally necessary to redistribute the carbohydrate content of the diet when a patient is changed from a soluble to a protamine insulin, so that the carbohydrate is approximately equally divided at the four meals. Owing to its slow rate of absorption, protamine insulinate and protamine zinc insulin suspension are useless for the treatment of hyperglycæmic coma. To summarise the use of these insulins, it may be said that no patient should change to them if he is well stabilised on soluble insulin. They are chiefly of value if with soluble insulin there is nocturnal hyperglycæmia with early morning glycosuria. Protamine zinc insulin is useful when only one injection of insulin is desired by patients who require two or more daily injections of soluble insulin.

### Low Carbohydrate Diets

Low carbohydrate diets, with a carbohydrate content of about 100 to 140 G., have a very definite place in the treatment of diabetes. The method of preliminary starvation, followed by a series of graduated diets, will often enable a patient to be stabilised on an adequate diet without the use of insulin. Further, in some cases the patient's urine can be rendered sugar-free without preliminary starvation and without insulin by the use of an intermediate low carbohydrate diet. He can then be worked up to an adequate diet, with or without insulin, according to his tolerance. Vegetables and fruits are classified according to their carbohydrate content as follows:—

*Vegetables A* (0 to 5% available carbohydrate) include: Artichokes (boiled), asparagus tips (boiled), French beans (boiled), runner beans (boiled), broccoli tops (boiled), Brussels sprouts (boiled), cabbage (boiled), carrots (boiled), cauliflower (boiled), celery (raw or boiled),

cucumber (raw), leeks (boiled), lettuce (raw), marrow (boiled) mustard and cress (raw), mushrooms (raw or fried), onions (boiled), radishes (raw), seakale (boiled), spinach (boiled), spring greens (boiled), swedes (boiled), turnips (boiled), watercress (raw), apples (stewed), black berries (stewed), cherries (stewed), cranberries (stewed), currants (stewed), gooseberries (stewed), raspberries (stewed), rhubarb (stewed), and tomatoes (fried or raw)

*Vegetables B* (5 to 10% available carbohydrate) include Beetroot (boiled), broad beans (boiled), spring onions (raw), peas (boiled), apricots (raw), blackberries (raw), currants (raw) damsons (raw) green figs (raw), gooseberries (raw), peaches (raw), plums (raw), raspberries (raw), strawberries (raw), grapefruit and oranges

*Vegetables C* (10 to 15% available carbohydrate) include Parsnips (boiled), apples (raw), cherries (raw), greengages (raw), nectarines (raw) and pears (raw)

*Vegetables D* (15 to 20% available carbohydrate) include Baked beans (tinned), butter beans (boiled), haricot beans (boiled), lentils (boiled), dried peas (tinned), potatoes (boiled), banana, grapes, dried peaches (stewed) and dried prunes (stewed)

*Vegetables E* (over 20% available carbohydrate) include Dried figs (stewed), dates and chestnuts

*Diet 1* Breakfast: Veg A, 3 oz, Egg, 1, Tea Lunch Veg A, 2 oz, Bovril, 10 oz, Water Tea Veg A, 2 oz, Tea Dinner Veg A, 3 oz, Egg, 1, Bovril, 10 oz, Water C 10 G, P 17 G, F, 12 G Calories = 216

*Diet 2* Breakfast: Veg A, 3 oz., Egg, 1; Butter,  $\frac{1}{2}$  oz, Energen bread, 1 roll; Tea Lunch. Veg A, 3 oz, Egg, 1, Bovril, 10 oz, Water Tea: Veg A, 3 oz, Egg, 1, Tea Dinner Veg A, 3 oz, Egg, 1, Bovril, 10 oz, Butter,  $\frac{1}{2}$  oz, Meat, 1 oz, Energen bread, 1 roll, Water C 16 G, P 39 G, F 40 G Calories = 580

*Diet 3* Breakfast. Veg A, 3 oz, Egg, 1, Butter,  $\frac{1}{2}$  oz, Energen bread, 1 roll, Tea, Bacon, 1 oz, Oatmeal,  $\frac{1}{2}$  oz, Milk, 2 oz Lunch. Veg A, 3 oz, Egg, 1, Bovril, 10 oz, Water, Butter,  $\frac{1}{2}$  oz, Cheese,  $\frac{1}{2}$  oz., Meat, 3 oz Tea Veg A, 3 oz, Egg, 1, Tea, Butter,  $\frac{1}{2}$  oz Dinner: Veg A, 3 oz, Egg, 1, Butter,  $\frac{1}{2}$  oz, Energen bread, 1 roll, Fish, 2 oz, Water C 28 G P 74 G, F 81 G Calories = 1,137

*Diet 4* Breakfast. Veg A, 3 oz, Egg, 1, Butter,  $\frac{1}{2}$  oz, Energen bread, 1 roll, Tea, Bacon, 1 oz, Oatmeal, 1 oz, Milk, 3 oz, Cream,  $\frac{1}{2}$  oz, Lunch: Veg A, 3 oz, Egg, 1, Bovril, 10 oz., Water, Butter,  $\frac{1}{2}$  oz, Cheese,  $\frac{1}{2}$  oz, Meat, 4 oz Tea. Veg A, 3 oz, Egg, 1, Tea; Butter,  $\frac{1}{2}$  oz Dinner. Veg A, 3 oz, Egg, 1, Butter,  $\frac{1}{2}$  oz, Energen bread, 1 roll, Fish, 2 oz., Water, Meat, 1 oz, Milk, 1 oz, Coffee C 41 G, P 91 G, F 94 G Calories = 1,374

*Diet 5* Breakfast: Veg A, 3 oz, Egg, 1, Butter,  $\frac{1}{2}$  oz, Tea, Bacon, 2 oz, Oatmeal, 1 oz., Milk, 3 oz, Cream,  $\frac{1}{2}$  oz., Brown bread,  $\frac{1}{2}$  oz Lunch. Veg A, 3 oz., Egg, 1, Bovril, 10 oz; Water, Butter,  $\frac{1}{2}$  oz, Cheese,  $\frac{1}{2}$  oz, Meat, 4 oz Tea. Veg A, 3 oz, Egg, 1, Tea, Butter,  $\frac{1}{2}$  oz., Energen bread, 2 rolls, Cream,  $\frac{1}{2}$  oz Dinner: Veg A, 3 oz., Egg, 1; Butter,  $\frac{1}{2}$  oz., Fish, 3 oz.;

Water; Meat, 2 oz.; Milk, 1 oz.; Brown bread,  $\frac{1}{2}$  oz.; Coffee. C. 54 G., P. 109 G., F. 115 G. Calories = 1,687.

*Diet 6.* Breakfast: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Bacon, 2 oz.; Oatmeal, 1 oz.; Milk, 2 oz.; Cream,  $\frac{1}{2}$  oz.; Tea. Lunch: Veg. A., 6 oz.; Meat, 3 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Cheese, 1 oz.; Water. Tea: Veg. A., 6 oz.; Butter,  $\frac{1}{2}$  oz.; Energen bread, 1 roll; Milk, 1 oz.; Tea. Dinner: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Fish, 3 oz.; Water; Meat, 3 oz.; Cream,  $\frac{1}{2}$  oz.; Coffee. C. 74 G., P. 119 G., F. 118 G. Calories = 1,834.

*Diet 7.* Breakfast: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Bacon, 2 oz.; Oatmeal, 1 oz.; Milk, 3 oz.; Cream, 1 oz.; Tea. Lunch: Veg. A., 6 oz.; Meat, 3 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Cheese,  $1\frac{1}{2}$  oz.; Water. Tea: Veg. A., 6 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Milk, 1 oz.; Tea. Dinner: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white), 1 oz.; Fish, 3 oz.; Water; Meat, 3 oz.; Cream,  $\frac{1}{2}$  oz.; Coffee. C. 89 G., P. 125 G., F. 132 G. Calories = 2,044.

*Diet 8.* Breakfast: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Bacon, 2 oz.; Oatmeal, 1 oz.; Milk, 3 oz.; Cream, 1 oz.; Tea. Lunch: Veg. A., 6 oz.; Meat, 3 oz.; Potato,  $1\frac{1}{2}$  oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Cheese,  $1\frac{1}{2}$  oz.; Water. Tea: Veg. A., 6 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $\frac{1}{2}$  oz.; Milk, 1 oz.; Tea. Dinner: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white), 1 oz.; Fish, 3 oz.; Water; Meat, 3 oz.; Potato,  $1\frac{1}{2}$  oz.; Cream,  $\frac{1}{2}$  oz.; Coffee. C. 106 G., P. 126 G., F. 132 G. Calories = 2,116.

*Diet 9.* Breakfast: Veg. A., 12 oz.; Egg, 1; Butter,  $\frac{1}{2}$  oz.; Bread (white), 1 oz.; Bacon, 2 oz.; Oatmeal, 1 oz.; Milk, 3 oz.; Cream, 1 oz.; Tea. Lunch: Veg. A., 6 oz.; Meat, 3 oz.; Potato,  $1\frac{1}{2}$  oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white), 1 oz.; Cheese,  $1\frac{1}{2}$  oz.; Water. Tea: Veg. A., 6 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white), 1 oz.; Milk, 1 oz.; Tea. Dinner: Veg. A., 12 oz.; Milk, 1 oz.; Butter,  $\frac{1}{2}$  oz.; Bread (white),  $1\frac{1}{2}$  oz.; Fish, 3 oz.; Water; Meat, 3 oz.; Potato,  $1\frac{1}{2}$  oz.; Cream, 1 oz.; Coffee. C. 135 G., P. 131 G., F. 149 G. Calories = 2,405.

The carbohydrate foods may, of course, be interchanged according to taste, attention being paid to the carbohydrate values. Thus 1 oz. of vegetable B. can be substituted for 2 oz. of vegetable A., and 1 oz. of vegetable C. for 3 oz. of vegetable A. Further, 12 oz. of vegetable A. are equivalent to 4 oz. of vegetable B., or  $2\frac{1}{2}$  oz. of vegetable C., or  $1\frac{1}{2}$  oz. of vegetable D., or  $\frac{1}{2}$  to 1 oz. of vegetable E. Various condiments and sauces may also be used in small quantities to render the dish more attractive; beef tea and chicken broth may be substituted for Lemco and Bovril. Saccharin up to gr. 3 daily may be used for sweetening purposes.

### High Carbohydrate Diets

Good results have been obtained in many cases of diabetes with the use of high carbohydrate diets containing 200 to 300 G. carbohydrate. The fat content of the diet should not exceed 50 to 80 G. They are of

particular value in children, young adults, and when the disease is complicated by nephritis, peptic ulcer and pulmonary tuberculosis. The following is an example of a diet used for a severe case, the patient being a girl aged 14. The insulin dosage required in this case is protamine zinc insulin 56 units at night, and soluble insulin 10 to 15 units before breakfast.

7 a.m. 2 biscuits ( $\frac{1}{2}$  oz) Breakfast: Apple, 2 oz, Egg 1, Bread, 1 oz, Oatmeal (dry), 1 oz, Jam,  $\frac{1}{2}$  oz Milk, 6 oz, Sugar,  $2\frac{1}{2}$  level teaspoonfuls. Mid-morning: Bread, 1 oz or 3 biscuits ( $\frac{3}{4}$  oz) Lunch: Bovril, 10 oz, Lean meat,  $1\frac{1}{2}$  oz, Potato (cooked) 3 oz, Butter,  $\frac{1}{2}$  oz, Apple, 2 oz, Veg A, 3 oz, Bread 1 oz Tea Milk, 4 oz, Bread, 2 oz, Plain cake, 1 piece (2 oz), Jam,  $\frac{1}{2}$  oz, Banana 1, or Bread, 1 oz Supper: Fish,  $1\frac{1}{2}$  oz, Potato (cooked) 3 oz, Butter,  $\frac{1}{2}$  oz, Apple, 2 oz, Milk, 4 oz, Veg A, 3 oz, Bread, 1 oz, Bananas, 2, or Bread, 2 oz 10 p.m. Milk, 4 oz, Plain cake, 1 piece (2 oz) or 3 biscuits ( $\frac{3}{4}$  oz) C 309 G, P 81 G, F 68 G Calories = 2172

When the patient is on a satisfactory diet a careful search should be made for septic foci in the mouth, nose and throat, etc., and if found they should be eradicated, a chronically inflamed appendix or gall bladder may require removal, before the metabolic processes are properly stabilised.

*Diabetic Treatment in case of Operations* If an operation is required it should be performed under a local or a spinal anaesthetic or with the aid of gas and oxygen, ether and such basal anaesthetics as Evipan Sodium (hexobarbitonium sol B.P. Add.) or Pentothal Sodium which are administered intravenously. Chloroform should never be given. The patient should be stabilised as described above and not starved before the anaesthetic. He should have his last meal 3 to 4 hours before the operation. Two and a half hours before the anaesthetic he should be given 30 units of insulin and half an hour later dextrose oz 2 dissolved in water oz 6, by mouth. On recovery from the anaesthetic the patient is given dextrose oz 5 to 6 by mouth or intravenously during the first 24 hours. Thus dextrose oz 2 dissolved in water oz 6 may be given 3 times during the 24 hours, with 10 to 20 units of insulin injected half an hour before each dose. No insulin should be injected unless the blood sugar is above 120 mg per 100 c.c., or if the urine is sugar free. Four pints of fluid in all must be given during the first 24 hours, either by mouth or intravenously. The next day the patient will probably be able to take citrated milk feeds of 4 to 6 oz every 3 hours with dextrose oz 1 in three of the feeds. Half an hour before these dextrose feeds 10 to 20 units of insulin should be injected. The urine must be tested for sugar before each insulin injection. The patient is then worked up again through the diets, but the earlier ones can usually be omitted.

*Diabetes and Pregnancy* A diabetic woman can safely be allowed to become pregnant if she is properly treated. Further, pregnancy is more likely to occur in a diabetic who is under treatment with insulin than in one who is not being so treated. Still birth is common in diabetes, unless the disease is accurately controlled. The blood sugar of an infant

born of a diabetic mother is often low (4 to 22 mg. per 100 c.c.) and hypoglycæmic convulsions may occur which are quickly relieved by the administration of dextrose. During pregnancy there is a tendency to ketonuria. When the patient is in labour, milk, dextrose and insulin should be given as described under the treatment in the case of operations. A Cæsarean section is not necessary. During the puerperium there is a tendency to hypoglycæmia and the insulin dosage may be reduced.

*After Treatment.* When the diet and the necessary insulin dosage are determined, the patient can begin to look after himself, with monthly tests by the doctor of the blood and urine. He should be instructed how to give the insulin, how to sterilise the syringe and needles, and how to test the urine morning and evening; he should also be warned of the danger of hypoglycæmia, that he must always have a carbohydrate-containing meal within half an hour of a dose of insulin, that he should not take violent exercise while he is having injections, that he should carry sugar with him to eat, should early symptoms of hypoglycæmia occur (see p. 630). If he gets a feverish cold his doctor should be informed, as he will temporarily require more insulin. He must always guard against constipation. He may also be given a table showing how his standard diet can be varied.

#### Treatment of Coma

**Coma Due to Ketosis.** The following routine should be observed: Test a specimen of urine, obtained by catheter if necessary, for glucose and ketone bodies; remove a specimen of blood and send it to the laboratory in a citrate or oxalate tube for sugar analysis. The urine in diabetic coma usually contains albumin. If the urine contains much sugar, give 40 to 60 units of insulin subcutaneously and 2 oz. of dextrose in 1 pint water by mouth, or 20 mls of a 10% solution administered intravenously if the patient is not able to swallow. The patient must be kept warm with hot bottles or an electric lamp under a cradle. Take the blood pressure; if this is below 100 mm. Hg. systolic, dextrose should be given intravenously, 20 mls of a 10% solution. If the blood pressure is about 70 mm. Hg. systolic, it usually means that the blood is "sticky." In this case a pint of hypertonic saline 1.2% should be given intravenously; stimulants such as Coramine (nikethamidum B.P.Add.) 15 to 5 mls or strophanthin gr. 1/250 may also be given intravenously to combat central respiratory and circulatory failure, and to counteract peripheral circulatory failure ephedrine hydrochloride gr.  $\frac{1}{2}$  in 1 mil. ampoule and Pitressin 1 mil. (20 units) may be injected intramuscularly. If the blood sugar is high, about 400 to 500 mg. per 100 c.c., more insulin should be injected at once; i.e., about 1 hour after the first injection, 50 units of insulin are given subcutaneously and 3 oz. of dextrose in a pint of water by mouth, or 20 mls of a 10% solution intravenously. The bowels should be opened by an enema unless the patient is very collapsed. Insulin and dextrose injections should be repeated every 3 hours, a specimen of urine being tested immediately before an injection is given; if the sugar in the urine is only a trace, no

more insulin should be given without a further blood test as the patient may insensibly be wafted from hyperglycæmic to hypoglycæmic coma. If there is much sugar large doses of insulin are repeated 500 to 1,000 units or more in the 24 hours. On recovery from coma the patient should be given milk oz 6 to 8 every 3 hours with insulin 3 times a day in amounts sufficient to keep the urine nearly free from sugar, acetone bodies will reappear and to get rid of them dextrose should be given in addition oz  $\frac{1}{2}$  to 1 in the three feeds which follow the insulin injections. In about 24 to 48 hours the patient can be put on a No. 3 diet and the extra dextrose continued until the acetone bodies disappear from the urine, the insulin being adjusted to keep the urine sugar free. A careful search for a septic focus should now be made, and if possible it should be eradicated.

**Coma Due to Hypoglycæmia** The patient who is receiving insulin should always have with him some lumps of sugar and eat them at the earliest symptom. Hypoglycæmic coma can usually be relieved by the intramuscular injection of 1 ml. of *liq. adrenalin hydrochlor.* and the intravenous injection of 20 to 100 mls. of 10% dextrose in normal saline, which should be repeated every half hour if necessary. In some cases as much as 200 G. of dextrose are required. Alternatively if the coma is not very deep dextrose oz 2 in water oz 6 may be introduced into the stomach through a nasal tube. When the patient can swallow he should be given dextrose oz  $\frac{1}{2}$  by mouth every hour for several hours especially if the hypoglycæmia is due to a protamine insulin.

### Acidosis

Acidosis implies that there is a lowering of the alkali reserve (chiefly bicarbonates) in the blood. There is not necessarily an acidæmia i.e. increase in the hydrogen ion concentration of the blood. In ketosis such abnormal substances as  $\beta$  hydroxybutyric acid and acetone are present in the blood.

**Clinical Findings** *Acidosis is of importance in several conditions such as Diabetes this is described on p. 623. Unexplained pyrexia in children and possibly attacks of migraine and asthma and also recurrent cyclic vomiting may be due to acidosis, such children are abnormally intolerant of fat in their diet or of fat containing medicines such as cod liver oil. Acidosis in nephritis may occur in the acute or chronic variety. Preliminary purgation and starvation before a general anæsthetic predispose to subsequent acidosis, as manifested by severe vomiting, the patient may gradually become unconscious and die in a few days. Severe starvation leads to acidosis but toxic symptoms do not usually show themselves. Salicylates are sometimes given to children suffering from pyrexia on the assumption they are suffering from rheumatism, when in reality they are acidotic. This makes their condition worse. Carbon monoxide poisoning causes acidosis by interference with the oxygen supply of the tissues. In acute yellow atrophy of the liver and in pernicious vomiting of pregnancy, acidosis may occur probably due to the toxic effects on the liver. The symptoms vary to a certain degree with the predisposing causes, they include headache*

saintness, nausea, vomiting, abdominal pain, air hunger, neuritis and pyrexia.

**Differential Diagnosis.** This is effected by: 1. Examination of the urine for acetone bodies. 2. Determination of the alkali reserve of the blood. 3. Determination of the alveolar  $\text{CO}_2$  tension.

**Treatment.** *Prophylactic.* Acidosis can be prevented by ensuring a sufficiency of carbohydrate in the diet, together with insulin if required in diabetes to metabolise the carbohydrate. In cyclic vomiting in children, the milk should be reduced to  $\frac{1}{2}$  pint daily, no cream should be allowed and plenty of carbohydrates must be given. In addition sodium bicarbonate gr. 20 to 30 should be given twice daily in water. Patients should not be starved and purged before operations.

*Curative.* Dextrose must be given by mouth oz. 1 to 2 daily, or by rectum or intravenously, so that the glycogen content of the liver is maintained. Insulin can also be injected as in the treatment of diabetic coma (see p. 636). In the cyclic vomiting of children it is well to give rectal injections of 3 fl. oz. of normal saline containing 5% dextrose every 4 to 8 hours, as the child will probably be unable to retain anything by mouth.

### Alkalosis

Alkalosis is characterised by an increase in the available alkali reserves in the blood. In alkalemia the hydrogen ion concentration is lowered.

**Clinical Findings.** Alkalosis results from administration of massive doses of alkali, as in the treatment of gastric or duodenal ulcers, especially if there is gastric stasis or renal insufficiency, and in the alkali treatment of chronic nephritis with oedema. It may also be caused by persistent vomiting, due to obstruction at the pylorus or high up in the intestine. In hyperventilation and in anoxaemia associated with high altitudes or heart failure there is alkalemia with a fall in the alkali reserves. The chief symptoms are: Malaise, headache, giddiness, anorexia, vomiting, tetany and coma.

**Differential Diagnosis.** This can be confirmed by determining the alkali reserve of the blood, and the blood nitrogen figures are usually raised.

**Treatment.** This is described on p. 38.

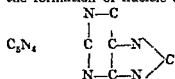
### Gout

**Definition.** A disease characterised by excess of uric acid in the blood, uratic deposits in tissues, and joint manifestations. Podagra implies gout in the foot and cheiragra gout in the hand.

**Etiology.** The cause is not known. There is possibly a metabolic error with regard to nucleoproteins, combined with a sensitiveness to certain protein substances, such as are contained in foods or bacteria, which precipitate an attack. Gout is often associated with asthma, urticaria or eczema, diseases due in some instances to a toxic idiopathy. Gout is not due solely to excess of uric acid in the blood, as much higher figures are obtained in such diseases as leukaemia, without any manifestation of gout. *Predisposing causes:* 1. Heredity: The gouty diathesis

is frequently noted in Europe, rarely in America 2 Age Usually middle age and after 3 Sex Males predominate 4 Food and alcohol Overeating, especially of meat, and overdrinking, especially of beer, port and sweet wines 5 Occupation Workers in lead and maltsters are prone to gout 6 Infection In teeth, tonsils, nasopharynx and the intestinal tract 7 Season Especially in the spring 8 Locality Chiefly in temperate zones, especially England and Germany, gout is rare in Scotland, probably due to a low incidence of beer and wine drinking An attack may be precipitated by trauma to a joint, by mental worries, by certain articles of diet which vary in different individuals, such as strawberries, white wine, port, etc., and by infection with micro-organisms Gout is much less common nowadays, presumably due to moderation in food and drink and the greater attention given to the eradication of septic foci

**Pathology** There is an excess of uric acid in the blood, the average amount in gout being 5 to 9 mg per 100 c.c. (normal 1 to 3 mg per 100 c.c.) Deposits of sodium mono urate are found in tissues which are comparatively avascular and rich in sodium Thus tophi ("chalk stones," composed mainly of urates) occur in the cartilage of the ear, eyelid, nose, in the olecranon and patella bursæ, around joints especially of the hands and feet and in tendons and ligaments The uric acid elimination in the urine is diminished for a day or so before an acute attack of gout, but increases during the attack Uric acid is derived from nucleoprotein, nucleoprotein is present in the cell nuclei of the tissues (endogenous source) and in the nuclei of foodstuffs (exogenous source) It may also be synthesised to a slight degree in the body from histidin and arginin Nucleoproteins in the body are metabolised with the formation of nucleic acid, a substance containing the purin ring



This is converted into adenine (amino-purin) and guanine (diamino purin), and by oxidation into hypoxanthine (oxypurin), xanthine (dioxypurin) and finally into uric acid (trioxypurin) It is now considered

probable that all the uric acid formed in man is not excreted, some being further katabolised in the blood

Post mortem examination of an affected joint shows whitish smears of urate on the cartilage, just under the surface, the palms of the hands may show white lines, the joints are deformed, tophi may be seen ulcerating through the skin Death usually results from complications due to cardio-vascular degeneration or chronic nephritis Thus myocardial degeneration, pericarditis, atheroma, arteriosclerosis, cerebral hæmorrhage, chronic nephritis or bronchitis may be found Uric deposits may be seen at the apex of the pyramids of the kidneys

**Clinical Findings** *Acute Gout* The patient is usually a middle-aged man, who may give a history of previous attacks or of digestive troubles For a few days before the attack he feels irritable, depressed and may have flatulent dyspepsia, acidity and constipation The attack usually begins during the night, the patient waking with severe pain of a burning boring character in the metatarso phalangeal joint of the big toe, in



the ankle or heel; he may shiver and sweat, and after several hours' agony fall asleep to awake in the morning and find the joint swollen, red, hot, tense and shiny. Post-operative gout is described appearing from a few hours to a few days after a surgical operation.

**On Examination:** During the attack the temperature is usually raised to about 100° to 102° F. There is œdema around the affected part, the neighbouring lymphatic glands may be enlarged and the veins engorged. The pain usually recurs every night for 2 or 3 nights, and the attack then passes off. The skin may desquamate over the joint, which feels numb. At times the attack begins during the day, or it may begin at night and last through the day. After the attack the patient often feels extremely fit. Other joints less frequently affected are those of the finger or thumb, the knee, wrist or elbow, but very rarely the hip or shoulder. Polyarthritic attacks are infrequent. The blood: During an attack this shows a polymorphonuclear leucocytosis of 20,000 to 25,000 per c.mm. The sedimentation rate of the red cells is considerably increased during the acute stage. The urine: Uric acid excretion falls before, and rises during the attack for 2 or 3 days.

**Differential Diagnosis.** Acute gout must be distinguished from acute rheumatism, suppuration around or in a joint, gonorrheal arthritis, infective arthritis, synovitis, an inflamed bunion, and tuberculous or syphilitic arthritis. The characteristics of acute gout are the typical appearances and history, the presence of tophi in the ears (see Fig. 62), etc., and a blood uric acid content of over 3.5, and often of 5 to 9 mg. per 100 c.c.

**Course and Complications.** A second attack may never occur, but usually it develops within a year, subsequent recurrences are more frequent. Complications include suppuration around tophi; cardio-vascular degeneration and chronic nephritis are often met with.

**Prognosis.** Gout is seldom fatal, unless associated diseases are present. Death is usually due to cardio-vascular or renal lesions.

**Treatment. Prophylactic.** When there is gout in the family, the precautions required are the eradication of all septic foci and moderation in eating and drinking.

**Curative.** During the attack: The leg or arm should be supported on a pillow, covered with wool and guarded by a cradle. Warm fomentations of lotio plumbi c. opio (B.P.C.) on lint, or of Sod. bicarb. oz. 1, tnc. opii fl. oz. 1 and water to a pint may be applied. A purge of calomel gr. 3, followed by mag. sulph. gr. 120 in the morning, should be given. A prescription of colchicum should also be ordered, such as Vin. colchic. (B.P. 1914) m. 20, pot. cit. gr. 30, mag. sulph. gr. 60, mag. carb. lev. gr. 10, aq. menth. pip. dest. ad fl. oz. 1. Fl. oz. 1 in hot water every 4 hours. When the bowels have been well opened, the magnesium sulphate is omitted and the medicine given 3 times a day. The patient should drink plenty of fluids, 4 to 8 pints in the 24 hours, hot water, hot weak tea, orangeade, or barley water, and he may also have toast, milk, bread and butter, and milk pudding. After the acute symptoms have subsided the diet is increased, fish and chicken being given and a course of Atophan (cinchophenum B.P.) may be



FIG 62 EAR SHOWING GOUTY TOPHI



FIG 63 ULCERATING TOPHI IN GOUT

begun This increases the elimination of uric acid One gr  $7\frac{1}{2}$  tablet is given twice a day for 3 days a week, for 4 weeks At the same time sod bicarb gr 60 should be given t d s p c Overdosage or idiosyncrasy may cause acute yellow atrophy of the liver, and as there is no absolutely safe way of giving cinchophen it is best avoided Equally good results, with less risk, are said to be obtained by the use of Sod salicyl gr 20, sod bicarb gr 30, syr aurant m 30, aq ad fl oz 1 Fl oz 1 t d s for 3 days a week for 4 weeks Sleep is best secured by paraldehyde m 30 capsules in doses up to m 120 to 240 All infective foci in the mouth should be eradicated

**Chronic Gout** Gout is considered to be chronic when an attack lasts for several weeks, or when attacks recur at short intervals Usually several joints are affected, permanent deformity ensues and the general health is impaired The tophi may ulcerate through the skin and suppuration occur around them (see Fig 63) There may be thickening of the olecranon and prepatellar bursæ The urine may contain a trace of albumin or of glucose Eczema is often present The tophi usually differentiate chronic gout from osteoarthritis, although the two conditions may be combined X ray examination of the hands may show "punched out" areas in the distal parts of the phalanges Fibrositis is sometimes a manifestation of chronic gout

**Treatment Diet.** The patient should avoid substances rich in purins, such as sweetbreads, liver, kidneys, brains, sardines, anchovies, turkey, pork, veal, beef, duck, goose partridge, bacon, rabbit, lamb meat soups and extracts, and lentils Purin poor substances are advisable, these include white bread, butter, cheese, eggs, rice, tapioca, milk, cereals, and green vegetables Fruits such as peaches, apples, pears, grapes cherries, and oranges may be given As the blood uric acid rises on a high fat diet, the amount of fat taken should be strictly limited Carbohydrates do not appear to be harmful Plenty of fluid, such as hot water, should be drunk on rising and retiring Tea coffee and cocoa may be taken in moderation It is better to avoid all alcohol A little "dry" wine or whisky may be allowed The bowels should be kept open regularly and a weekly dose of calomel gr 3 or blue pill (pil hydrarg B P) gr 4 should be given, followed by mag sulph gr 120 next morning Various medicines are of value, such as Pot iod gr 3, and guaiacol carbonate gr 5 in a cachet t d s, or Pot iod gr 3, in colchic (B P 1914) m 15, the hyoscyam m 30, infus columb rec 2d fl oz 1 Fl oz 1 t d s p c In some cases better results are obtained with a pill of Colchicine gr  $1/70$ , ext nuc vom gr  $\frac{1}{4}$ , ext hyoscyam gr  $\frac{1}{2}$ , lactose gr  $\frac{1}{2}$  t i d Periodic courses of sodium salicylate, as detailed above, aid in the elimination of uric acid Ulcerating tophi can be removed surgically through the adjacent healthy skin All septic foci should be eliminated

**Irregular Gout** (Suppressed or retrocedent gout) In some cases an acute attack of gout ceases suddenly, as may happen if the affected part is put into cold water The patient may become unconscious or die Death is probably not due to gout but to associated degeneration in other organs, such as the cardio-vascular or renal systems There is

no evidence that such a condition as gout in the stomach exists, many cases are due to such diseases as gall-stones, gastritis or appendicitis, and this also applies to the so-called gouty iritis which is considered to be infective in origin.

### Obesity

#### (*Lipomatosis Universalis*)

**Definition.** Excessive generalised deposition of fat in the body.

**Physiology and Pathology.** The diet of an average healthy male, who is not doing heavy muscular work, is composed of about 500 G. of carbohydrate, 100 G. of protein and 100 G. of fat, and yields 3,300 calories. In order to maintain a steady weight the energy output must balance this intake. The energy expended is made up of basal metabolism, metabolism due to muscular activity, and possibly metabolism resulting from the specific dynamic action of food. As a diet of 3,300 calories yields more energy than is necessary for physiological requirements, the metabolic processes are presumably raised in individuals who do not put on weight. Obesity may be:—

1. Developmental. This is usually a hereditary condition.
2. Nutritional or exogenous. This may be due to overeating, overdrinking and lack of exercise. In some cases there is water retention in the tissues. It is not known whether some individuals absorb more of their food than do others.

3. Metabolic or endogenous. This may result from endocrine disturbances, such as hypothyroidism (see p. 654), hypopituitarism (see p. 671), hyperadrenia (see p. 663) and deficiency of secretion from the ovaries (as at the climacteric) or from the testicles (as in eunuchs).

**Clinical Findings.** The weight of the patient is usually several stones above the normal for the age and height, the disposition is generally calm and cheerful, the only complaint being due to the personal appearance and discomfort of the excess of fat, which may also cause dyspnoea from cardiac embarrassment. Investigation of cases of obesity involves first an inquiry into the history as regards heredity, diet and exercise. An estimation should be made of the basal metabolic rate, sugar tolerance and water elimination, and special tests performed with respect to the ductless glands (see p. 670), such as X-rays of the skull, etc. Unless the obesity is due to hypothyroidism, the basal metabolic rate is usually within normal limits. The sugar tolerance is usually diminished in cases of marked obesity, the curve falling within normal limits as the weight is reduced by dieting.

**Course and Complications.** The condition is often slowly progressive, and complications may occur, such as fatty infiltration or degeneration of the heart, diabetes mellitus and chronic bronchitis.

**Prognosis.** Obesity tends to lower the expectation of life, owing to the liability to complications; further, severe illnesses and abdominal operations are less well borne.

**Treatment.** *Prophylactic.* Where a hereditary tendency exists, a regular check should be kept on the weight. Any increase should at once be countered by a dietetic restriction or increase in exercise. As a

man weighing 70 kg will only use up 140 calories on walking  $2\frac{1}{2}$  miles in an hour, it can be seen that reduction of diet is of greater importance than an increase of exercise in keeping the weight within bounds

*Curative* In developed cases of exogenous obesity the weight should be gradually reduced by about 2 to 3 pounds a week. The caloric value of the patient's normal diet should first be determined, it will often be found to be high, over 4,000 calories. Sugar, potatoes, cakes, sweets, wines and beer should then be eliminated and bread reduced. If this does not produce sufficient loss in weight, the patient should be placed on a special diet containing about 500 to 1,000 calories. It should contain about 1 G of protein per kg of bodyweight, and not too much fat or severe acidosis is liable to develop. The following diet (Ruthin Castle) of about 1,000 calories, containing approximately protein 80 G, fat 38 G, and carbohydrate 77 G may be used

*Breakfast* Grilled fish (4 oz), or grilled fish ( $2\frac{1}{2}$  oz) and egg (1), or bacon ( $2\frac{3}{8}$  oz) and tomatoes (3 oz), or cold ham (2 oz), or bacon ( $\frac{1}{2}$  oz) and egg (1) or eggs (2) boiled or poached. Toast ( $\frac{1}{2}$  oz) Butter ( $\frac{1}{8}$  oz) Sugarless marmalade ( $\frac{1}{2}$  oz) Milk ( $1\frac{1}{2}$  oz) Tea

*Lunch* Roast beef or mutton, hot or cold (2 oz), or grilled steak, boiled beef, grilled chop, boiled mutton, roast turkey or chicken ( $2\frac{1}{2}$  oz), or veal (3 oz), or grilled sweetbreads (3 oz) with bacon ( $\frac{1}{2}$  oz), or liver ( $3\frac{1}{2}$  oz) Green vegetables (4 oz) or salad (4 oz) Stewed fruit (3 oz) or fresh fruit (4 oz) Toast ( $\frac{1}{2}$  oz) Butter ( $\frac{1}{8}$  oz)

*Dinner* Fried fish ( $2\frac{1}{2}$  oz) or meats as at lunch, or eggs (2) boiled, poached or made into an omelette. Vegetables, fruit, toast and butter as at lunch. Toast may be increased to 1 oz, if the vegetables are omitted. Tea

*Supper* Water biscuits (2) Milk (1 oz) Tea

In severe cases of obesity, where an adult weighs over 18 st a diet of 500 calories will be required if weight is to be lost at the rate of 3 to 4 lb a week. When the weight has been reduced by 5 or 6 st the 1,000 calories diet can be substituted. A 500 calories diet containing protein 36 G, fat 16 G and carbohydrate 56 G, is as follows —

*Breakfast.* Egg (1) boiled or poached, or lean ham (1 oz) Vita Weat or Ryvita biscuits (2) Tomatoes (4 oz) or Ryvita biscuit ( $\frac{1}{2}$ ) Milk (1 oz) Tea or coffee

11 a.m. Orange (3 oz) or its equivalent (see below)

*Lunch* Clear soup, if desired. Lean meat (2 oz), or steamed white fish (3 oz) and butter ( $\frac{1}{4}$  oz) Green vegetables (6 oz) Orange (3 oz) or its equivalent (see below)

*Tea* Green salad as desired (lettuce, endive, watercress, mustard and cress, and celery) Vita Weat or Ryvita biscuit (1) Milk (1 oz) Tea

*Dinner* Egg (1) poached or boiled, or lean ham (1 oz) or sardines with no oil (1 oz), or cheese ( $\frac{3}{4}$  oz) Green salad as desired. Vita Weat or Ryvita biscuits (2) Orange (3 oz) or its equivalent (see below)

The following are equivalent to orange 3 oz — Fresh pineapple 2 oz, raw apple, pear or grapefruit 3 oz, raw cherries  $3\frac{1}{2}$  oz, stewed apple, pear or cherries 4 oz, strawberries  $4\frac{1}{2}$  oz, raw redcurrants 5 oz, stewed

greengages 6 oz., raw blackcurrants or raspberries 7 oz., stewed damsons 7 oz., melon  $7\frac{1}{2}$  oz., stewed raspberries or plums or raw tomatoes 8 oz., stewed blackcurrants 10 oz., stewed gooseberries 13 oz., 1 Vita Weat biscuit.

In addition to this diet the patient should take 3 pints of fluid in the 24 hours, including a glass of hot water on rising and retiring. Saccharin can be used for sweetening. Exercise should also be taken, a walk of a mile or more daily or simple exercises such as contracting the trunk and abdominal muscles. The Bergonié chair forms a convenient method of provoking skeletal muscular contractions in those unwilling to exercise normally. In obesity due to hypothyroidism, thyroideum gr.  $\frac{1}{4}$  to 1 can be given daily and gradually increased. Salyrgan (mersalylum B.P.Add.) (see p. 228), a salt-poor diet and restriction of fluid intake to 35 or 40 oz. in the 24 hours should be used for excess of weight due to water retention.

### Localised Lipomatosis

Localised deposits of fat may occur in the following conditions:—

1. Lipoma, single or multiple. These should be removed if growing rapidly owing to the risk of sarcomatous changes.
2. Localised obesity, the fat accumulating over the hips or abdomen. Vibration and massage are useful in these cases. Gluteal humps of fat are met with in Bushmen and Hottentots. They are analogous to the camel's hump and become flabby during starvation. The condition is known as steatopygy. Symmetrical excess of adipose tissue may form in the neck, constituting the "fat neck" of Madelung.
3. Adiposis dolorosa (see p. 672).
4. Pseudo-hypertrophic muscular dystrophy.

### Lipodystrophia Progressiva

A disease of children characterised by progressive wasting of the face, neck, arms, thorax and abdomen. The cause is not known, it has been suggested that it is due to a tropho-neurosis. The buttocks and legs are usually normal, but in some cases are the only parts affected. The wasting is due to loss of fat, the tone of the muscles being normal. The loss of fat is steadily progressive for periods up to 2 or 10 years. The general health is not impaired.

### Localised Lipodystrophy

Loss of subcutaneous fat occurs in some individuals in the arms or legs, in association with repeated injections of insulin. Local panatropy may also occur apart from insulin injections, with wasting of the subcutaneous tissues including muscles. It may be seen in the arm or leg and the cause is unknown.

### Ochronosis

**Definition.** A rare disease characterised by pigmentation of cartilage and skin, arthritis and urinary changes.

**Etiology.** There are three groups of cases: 1. Associated with alkaptonuria, an inborn error of metabolism, in which homogentisic acid

is excreted in the urine 2 Associated with chronic absorption of carbolic acid, used for dressing wounds, usually ulcers on the legs, for prolonged periods, such as 20 or 30 years Carboluria is usually present.

3 Occurring apart from either of these conditions

**Pathology** Melanin is deposited as yellow brown particles in the cartilage of the ears, nose, eyelids, trachea, bronchi and in the ligaments and fibrous tissues of the body Chronic osteoarthritis may be present in the large joints In alkaptonuria, homogentisic acid is passed in the urine, owing to incomplete metabolism of tyrosine

**Clinical Findings** The patient is usually an adult who seeks advice either on account of the pigmentation of the ears or face, or because of arthritis. A child may be brought to the doctor because his urine darkens on standing, or the patient may seek advice because he has been rejected for life assurance In the cases due to carboluria a history is usually obtained of a wound being treated with carbolic dressings for a prolonged period

**On Examination** The cartilaginous parts of the ears appear bluish, and a blue black area of pigmentation may be seen in the sclerotics The tendons of the hands are pigmented and the skin of the face and hands may be yellow brown or brownish black in patches Osteoarthritis may be found in the knees or other joints The urine may contain homogentisic acid, becoming dark on standing, staining the clothes and reducing Fehling's solution, or it may give the reaction for carboluria

**Differential Diagnosis** Alkaptonuria must be differentiated from glycosuria by chemical tests

**Course and Complications** Alkaptonuria is a congenital abnormality and persists through life

**Prognosis.** Alkaptonuria does not shorten life

**Treatment** *Prophylactic* Carbolic acid should not be used for dressings for prolonged periods

*Curative* If the ochronosis is due to carbolic acid, the dressings must be discontinued, in alkaptonuria the protein in the diet should not exceed 100 G a day

## Hæmochromatosis

(*Diabete Bronzé*)

**Definition** A disease characterised by pigmentation of the skin, fibrosis of the liver and pancreas, and glycosuria

**Etiology** Hæmochromatosis is probably due to an inborn error of iron metabolism Males predominate over females in the proportion of 10 to 1

**Pathology** Hæmosiderin is deposited in excess in nearly all the tissues, including the skin Hæmofuscin (iron free), which is related to melanin and contains sulphur, is deposited in the heart and intestinal muscle This does not result from increased blood destruction, but may be due to inability of the organs to excrete the pigment The liver, pancreas and spleen are cirrhotic and the glycosuria is considered to be secondary to the pancreatic changes (chronic interstitial pancreatitis)

## CHAPTER XI

### THE DUCTLESS GLANDS

#### THE THYROID GLAND

**Introductory** The follicles (vesicles or alveoli) of the thyroid gland contain colloid, secreted by the lining cuboidal epithelium. The active principle of the gland is present in the colloid, which is rich in iodine. Thyroxine has been obtained from the gland and also synthesised. It is probably an intermediate stage of the active principle and contains 65% of iodine. The active principle leaves the thyroid by the blood vessels and is possibly a combination of thyroxine and a peptide. One milligramme of thyroxine is said to raise the B.M.R. of man by 3%. A daily intake of 0.16 mg. of iodine is sufficient to maintain the thyroid function. The thyroid secretion is regulated by the thyrotropic hormone secreted by the anterior lobe of the pituitary.

#### Simple Goitre

(*Endemic and Sporadic Goitre Diffuse Parenchymatous Goitre or Colloid Goitre Nodular Goitre with Localised or Multiple Adenomata*)

**Definition** General or local enlargement of the thyroid gland without marked disturbance of thyroid function. The enlargement is neither due to inflammation nor to malignant disease.

**Etiology** There are two main theories as to the etiology of colloid goitre. 1. Deficiency of iodine in the diet or the drinking water. 2. Impurity of the water due to organisms or suspended calcareous matter. Possibly in goitrous water there is something which prevents the utilisation of iodine, as endemic goitre has been eradicated in some districts by giving pure water free from all iodine or by adding iodine to the water. **Predisposing causes** 1. Locality. Derbyshire and the Thames Valley in England, the Swiss Alps, Pyrenees, Maritime Alps, Himalayas, Rocky Mountains, etc. Air-borne iodine is present within three miles of the sea coast. 2. Age. Children and young adults. 3. Sex. Chiefly females and often associated with puberty, pregnancy and lactation. 4. Inter-marriage of near relations.

**Pathology** *Colloid Goitre* The gland is uniformly enlarged. The vesicles are distended with colloid and lined with atrophied epithelium. The iodine content is increased.

*Parenchymatous Goitre* The gland is uniformly enlarged. The vesicles contain less colloid than normal and the epithelium tends to be columnar shaped. The iodine is diminished.

*Nodular Goitre* Localised or diffuse adenomata may occur. The adenoma may develop from a foetal rest. The gland may be smaller than normal, with increased fibrous tissue. The adenoma may be solid or cystic, and hæmorrhages may be present in the gland. *Pressure of*



More common in females. 3. Hard work. 4. Absence of any previous goitre. 5. Locality. Districts free from endemic goitre. 6. Heredity. A thyrotoxic diathesis may be transmitted.

**Pathology.** The thyroid gland is enlarged and vascular with increased fibrous tissue. There is an increase in the alveolar epithelium which may project in columnar celled ridges nearly filling the lumen of the vesicles. The colloid is diminished and the iodine content of the gland low. Hyperplastic lymph nodes are constantly present in the gland. Adenomata or cysts may be present. Rienhoff has shown that after iodine treatment the vesicles contain more colloid, the lining epithelium becomes flat and the hyperplastic gland reverts to the resting colloid state. The thymus is often enlarged, and the cervical lymph glands are enlarged. The cause of the ocular prominence is unknown. It may be due to retro-orbital vascular dilatation, to lymphocytic infiltration, oedema, or fibrosis of the ocular muscles, or to the action of the pituitary thyrotropic principle. There is no evidence that it is due to excess of retro-orbital fat, to stimulation of Muller's muscle, which is a vestigial structure in man, or to sympathetic stimulation. The pupil is not dilated in Graves' disease and exophthalmos may occur when the cervical sympathetic is paralysed due to long-standing syringomyelia.

**Clinical Findings.** The patient sometimes gives a history of a shock or intense worry which is followed by symptoms of nervousness, irritability and disturbed sleep. The onset may be quite sudden, the patient noticing that one or both of the eyes is prominent or that the neck suddenly swells. Other symptoms include loss of weight, lassitude, nervousness, palpitations, shortness of breath, moisture of the skin, especially on the palms of the hands, and hair falling out. The patient usually feels worse in hot weather, the bowels may be relaxed and attacks of vomiting occur. Acute abdominal pain may simulate that produced by appendicitis. In some cases there are symptoms of diabetes insipidus, presumably due to pituitary disturbance. In the early stages there may be menorrhagia which is followed later by amenorrhœa.

**On Examination:** The patient is usually rather thin and has a characteristic startled appearance owing to prominence of the eyes. The skin is moist and may be flushed, especially on the face and over the neck and manubrium sterni. There may be pigmentation, especially on the face, arms and trunk, with leucodermic patches. Carotid pulsation is seen in the neck. The typical signs of Graves' disease are: 1. Enlargement of the thyroid gland. The swelling is moderately soft and uniformly enlarged, and if the patient raises up the head while lying down the swelling largely disappears. Irregularities may be caused by a cyst or adenoma. A systolic murmur is heard over the lateral lobes of the gland. 2. Tachycardia. The pulse rate is usually over 120 when the patient is at rest. 3. Tremors. Fine tremors of the fingers are seen when the hands are held out. 4. Eye signs. These include exophthalmos, the prominence of the eyes being generally bilateral, rarely unilateral (see Fig. 64). Exophthalmos is present in



FIG 64 UNILATERAL EXOPHTHALMOS IN GRAVES  
DISEASE



FIG. 65. ACROMEGALY.

about 95% of cases Corneal ulceration may occur *Von Graefe's sign* When the patient looks down, there is lagging of the upper lid *Stell wag's sign* Blinking is infrequent *Moebius sign* Lack of convergence of the eyes when the patient looks at an object which is brought near to her *Joffroy's sign* When the patient looks up, the forehead is not wrinkled *Dalrymple's sign* Wideness of the palpebral fissure, due to retraction of the upper lid The Merseburg triad of signs, described by Basedow in a patient living in Merseburg consists of goitre, exophthalmos and tachycardia The heart The impulse is forcible and the apex may be displaced a little outwards and downwards There may be irregularity due to premature systoles or auricular fibrillation with or without congestive heart failure Blood pressure This is raised in about 10% of cases When raised, there is an increase of the pulse pressure over the normal average of 40, e.g., systolic 180 and diastolic 100 The electrocardiogram This is normal in over 30% of cases, and flat or inverted T waves are the commonest abnormality The blood Lymphocytosis has been described but is not characteristic The sugar tolerance is often diminished although the resting blood sugar may be normal The blood iodine The total blood iodine normally amounts to 8 to 13  $\gamma$  per 100 c.c. ( $\gamma = 0.001$  milligramme) The iodine is present as an alcohol soluble fraction (7 to 12  $\gamma$ ) and an alcohol insoluble fraction (1 to 4  $\gamma$ ) In untreated Graves' disease the alcohol soluble iodine is usually normal, but the alcohol insoluble fraction is high (18 to 36  $\gamma$ ) After treatment with Lugol's iodine solution the alcohol soluble blood iodine is raised, the alcohol insoluble fraction is diminished, but not necessarily in proportion with the fall in the B.M.R. After thyroidectomy there is usually a slight decrease in the insoluble iodine The urine There may be glycosuria after a carbohydrate rich meal but often the renal threshold is raised so that no glycosuria occurs when the blood sugar reaches the normal leak point of 180 mg. per 100 c.c. Marked polyuria, resembling that of diabetes insipidus is noted in some cases Decalcification of bones is very constantly met with and calcium balance experiments show an increased loss both of calcium and phosphorus in thyrotoxicosis The basal metabolic rate is increased to a varying degree, usually between +20% and +60% A guide can be obtained by using Read's formula  $B.M.R. = 0.73 (P.R. + 0.74 P.P.) - 72$ , where P.R. = the resting pulse rate and P.P. the resting pulse pressure, i.e., the difference between the systolic and diastolic pressures The result is a positive or negative figure which is the B.M.R. expressed as a percentage Thus, if the answer is +40, the B.M.R. is +40%

**Differential Diagnosis** A typical case presents no difficulty In an early case there may be unilateral exophthalmos, when Graves' disease has to be differentiated from other causes such as a retro orbital tumour, or a general disease such as chloroma or xanthomatosis Here a determination of the B.M.R. is a valuable aid to the clinical findings Exophthalmic ophthalmoplegia is a condition associated with past or present thyrotoxicosis Individual movements of the eye, especially elevation are affected rather than individual muscles It may follow

vasodilatation, loss of weight, sweating, pigmentation, a tendency to diarrhoea and nervousness and a slight degree of staring of the eyes. The diagnosis is confirmed by finding a raised B.M.R. In some cases the B.M.R. is normal or only slightly raised, but after 14 days' iodine treatment it falls, showing that it was above the patient's normal reading.

**Treatment.** Lugol's iodine solution, given as for Graves' disease (see p. 652), usually produces improvement. This is often transitory or insufficient, and a partial thyroidectomy is generally required.

### Toxic Adenoma

(*Secondary Graves' Disease. Secondary Thyrotoxicosis*)

**Definition.** A disease characterised by the presence of one or more adenomata in the thyroid gland, later followed by thyrotoxic symptoms.

**Etiology.** The proliferation of the adenoma may be due to deficiency of iodine.

**Pathology.** Proliferation of the lining epithelium of the adenoma does not usually occur. The follicles contain colloid, or the adenoma may be filled with spheroidal cells conforming to the foetal type.

**Clinical Findings.** The patient is usually an adult over the age of 35, who has had a goitre for some years. Symptoms of hyperthyroidism then appear insidiously, as described on p. 649.

**On Examination:** The noteworthy features are the presence of an adenoma in the thyroid, the absence of exophthalmos and acute nerve crises, and the liability to auricular fibrillation. The other signs are as for Graves' disease (see p. 650), but the eye signs are not present. It should be noted that the supporters of the view that primary and secondary thyrotoxicosis are one and the same disease state that exophthalmos is rare in toxic adenoma owing to the later age incidence of the onset.

**Differential Diagnosis.** There is sometimes difficulty in distinguishing secondary from primary Graves' disease. If the adenoma is small or retrosternal it may not be palpable, and a clear-cut history of goitre antedating by several years the thyrotoxic symptoms will not be obtained.

**Course and Complications.** The course is typically progressive without remissions. Myocardial degeneration is common in untreated cases.

**Prognosis.** This depends upon the treatment. If recognised early and treated adequately there is good hope of permanent recovery, without recurrence after operation.

**Treatment.** Medical treatment is not successful. Iodine usually aggravates the symptoms, and should not be given for more than 2 weeks. The adenomatous tissue should be removed by operation.

### Hypothyroidism

#### Cretinism

(*Primary or Congenital Hypothyroidism*)

**Definition.** A disease resulting from congenital deficiency of thyroid secretion.

**Etiology** The cause is unknown. Cretinism may be associated with deficiency of iodine in the diet of the mother during pregnancy. It is more common in goitrous districts (endemic cretinism) the parents being goitrous, but it also occurs sporadically, especially in England, when the parents rarely have a goitre. The sex incidence is about equal.

**Pathology** The thyroid gland may be absent (athyreosis), or small and undeveloped, and show atrophy of the vesicular epithelium, with overgrowth of the connective tissue. Adenomata containing little colloid may be present in the gland of endemic cretins.

**Clinical Findings** The cretin does not develop normally from birth. The features which attract attention are delay in growth, intelligence and movement. The teeth may erupt late, and constipation is often troublesome.

**On Examination** A typical cretin presents a somewhat bloated appearance with lack of expression. The hair is scanty, the forehead low, the skin thick and dry, the nose flat, the tongue may be large, and the hands are podgy. The abdomen is prominent, and an umbilical hernia may be present. The thyroid gland may be difficult to feel or may be enlarged. The intelligence is of a very low grade, the child being practically an idiot, but quiet and easily managed. The pulse is slow, the temperature subnormal and the basal metabolic rate low (such as  $-40\%$ ). X-ray examination may reveal delay in ossification of the bones, but there is no overgrowth of the epiphyseal cartilage.

**Differential Diagnosis** In infancy the condition must be diagnosed from Mongolism (see p. 676), congenital or acquired mental deficiency, such as follows encephalitis lethargica, or other varieties of dwarfism. In Mongolism the appearances are characteristic. With congenital mental deficiency the baby may appear normal, but difficulty may be noticed in swallowing solid food or in sitting up, or there may be a squint. In dementia following encephalitis lethargica the child is often excitable, resembling an animal.

**Course and Complications** The course depends upon efficient treatment and the date of its commencement. An untreated cretin becomes an unintelligent dwarf who is often a deaf mute and lacks sexual power. If not treated for some years, but little improvement can be expected, especially as regards mentality. Complications are usually due to intercurrent disease.

**Prognosis** This is good if the condition is recognised and treated early, but thyroid extract will probably be needed during the whole of life and the mentality is usually, but not invariably, subnormal. The response to thyroid treatment is not so good in endemic cases.

**Treatment** *Prophylactic* In goitrous districts expectant mothers should be given sodium iodide gr. 1 t.d.s.

*Curative* The initial dose of thyroid extract for a cretin should always be small, such as thyroideum gr.  $\frac{1}{4}$ , increasing to gr. 1 or 2 t.d.s., according to the age of the child and the response obtained. Sufferers from thyroid deficiency are much more susceptible to thyroid extract than are normal individuals. Thyroid treatment should be discontinued temporarily during any intercurrent infection. The child should be

weighed every week. An operation may be required to remove an adenoma causing pressure.

Toxic symptoms due to overdosage include tachycardia, sweating, vomiting, diarrhoea, restlessness and loss of weight. Should these appear, the thyroid extract must be stopped, and, when the symptoms have passed off, the treatment should be started again with a smaller dose.

### Myxœdema

#### (Secondary or Acquired Hypothyroidism)

**Definition.** A disease due to acquired deficiency of thyroid secretion.

**Etiology.** The cause is unknown, except in those cases resulting from too extensive removal of the gland (cachexia strumipriva) or as a sequel of lymphadenoid goitre (see p. 658). *Predisposing causes:* 1. Age: Usually between 30 and 60. 2. Sex: More common in women. 3. Heredity: Myxœdema may run in families.

**Pathology.** The thyroid gland is small. The vesicles are few in number and fibrous tissue is present. In some instances there is a colloid goitre. The skin shows an increase of subcutaneous connective or fatty tissue, but there is no œdema, nor is mucin present.

**Clinical Findings.** The patient is usually an adult over the age of 40, who notices the gradual onset of such symptoms as lack of energy, torpor, increase in weight, deafness, supra-clavicular swelling, loss of hair, dryness of the skin, sensibility to cold, and constipation. There may also be pains in the muscles of the arms or legs near the joints, and a painful swelling may occur in a large joint such as the knee. The patient feels better in hot weather. A few cases with an acute onset have been recorded.

**On Examination:** The face is somewhat round and expressionless, the skin has a yellowish tinge, but is red over the malar bones. The eyelids may be swollen and so semi-closed. The lips may be swollen and the voice guttural. The hair is dry and rather scanty, and the outer parts of the eyebrows are deficient. The tongue may be enlarged and speech and cerebration slow. The thyroid gland is usually small, the skin is dry, the hands are rather clumsy, and although the arms and legs appear swollen, there is no pitting on pressure. Supra-clavicular pads of thickened subcutaneous tissue are characteristic. The heart may be dilated and the arteries thickened, but the blood pressure is not usually low, and may be high. The pulse is slow, the temperature subnormal, the basal metabolic rate is reduced to about — 30%, and the sugar tolerance increased. The blood cholesterol is raised and varies inversely with the B.M.R. A macrocytic or microcytic anæmia may occur. The urine may contain a trace of albumin. Menstruation may be irregular if the disease occurs before the climacteric.

**Differential Diagnosis.** The appearances of a typical case are characteristic. Obesity due to other causes must be excluded (see p. 642). The patient's relatives may think she is suffering from "nerves," or that she is a *malade imaginaire*. The absence of true œdema differentiates the swelling of the legs from that caused by

cardiac or renal disease. Swelling of the large joints may suggest osteoarthritis due to other causes (see p 618). Some cases have been mistaken for essential hypertension.

**Course and Complications** The condition is usually slowly progressive, but arrest may occur at any stage apart from treatment. There may be marked mental deterioration. Complications are due to intercurrent infections.

**Prognosis** There is usually marked improvement with treatment.

**Treatment** Thyroidum should be given beginning with gr  $\frac{1}{4}$  and increasing gradually to gr 2 or more t i d until the condition is relieved when the minimal dose required to maintain the improvement must be worked out. Care must be taken to give small doses, or palpitations, angina or cardiac failure may ensue. The return of the blood cholesterol to normal usually indicates that a sufficiency of thyroid is being given although the B M R may still be below normal. The urinary output is usually increased by the thyroid treatment. Constipation should be treated by attention to the diet, and the use of laxatives if necessary (see p 62).

### Masked Hypothyroidism in Adults

In masked hypothyroidism the patient presents no clinical features of myxœdema, but the B.M.R. is low and the symptoms are relieved by thyroid extract. Examples are afforded by certain cases of angina of effort, secondary amenorrhœa and dermatomyositis.

### Acute Thyroiditis

Acute thyroiditis is uncommon, and is more likely to affect a patient who has a goitre than one whose thyroid gland is normal. Young people are chiefly affected. It may complicate influenza, scarlet fever, typhoid fever, puerperal septicæmia, etc., or occur apparently spontaneously. The thyroid often swells suddenly with pain, dyspnoea and dysphagia. There is some fever and at times there are rigors. Unless abscesses form the swelling usually disappears in a few days. Cold applications should be applied to the neck, and sedatives, such as pot brom gr 10 to 15 given t i d. If there is suppuration an operation should be performed under general anaesthesia.

### Riedel's Disease

#### (Eisenhardt Strumitis)

A portion of the thyroid gland becomes extremely hard, like iron, suggesting a malignant tumour. The surface of the gland, however, is smooth and lymphatic glands are not involved. The change is a chronic inflammatory one, with fibrous tissue formation. The fibrous tissue in the affected portion of the gland invades surrounding structures such as the trachea and cervical muscles, and the carotid sheath. Men and women are equally affected, and it may occur in young people. There are often symptoms due to pressure on the trachea, œsophagus or recurrent laryngeal nerve, and pain may be referred to the ear. These



symptoms may be out of proportion to the size of the tumour. The skin does not adhere to the swelling. Owing to the difficulty in distinguishing it from a malignant tumour, removal by operation is advisable, but complete removal may be impossible on account of its adhesion to surrounding structures.

### Lymphadenoid Goitre

The cause of this condition is unknown. Women over the age of 45 are chiefly affected. The thyroid is firm and infiltrated with lymphocytes and the normal colloid is absent. The gland is uniformly enlarged and various pressure effects may be noticed. Myxœdema may ensue. *Treatment consists in the administration of thyroid extract for the deficiency symptoms, and operative removal to relieve pressure effects.*

### Tumours of the Thyroid

Simple tumours include an adenoma, also a fibroma or papilloma, which are rare. A teratoma is uncommon. A parathyroid tumour may be situated in the thyroid and resemble clinically a thyroid adenoma. Carcinoma is not very uncommon. It is usually a primary growth, but may be secondary to a tumour in the breast, tongue, or stomach, or the gland may be involved by a direct spread from carcinoma of the œsophagus. The characteristic features are the stony hardness of the tumour, its tendency to adhere to the skin, trachea or larynx, and the pressure symptoms due to deviation of the trachea, compression of the œsophagus or involvement of adjacent nerves, such as the recurrent laryngeal and sympathetic. Pain may be felt in the ear. Secondary deposits are very liable to form in glands, the lungs, bones, the liver, the eyes and in the skin. The diagnosis is often very difficult, but malignancy should be suspected if a tumour grows rapidly after a period of inactivity, if it becomes very hard, and if in addition toxic symptoms develop. The early onset of dysphagia is also very suggestive. Treatment is surgical, but the prognosis is usually hopeless. A secondary deposit in bone can give rise to hyperthyroidism after thyroidectomy. Its removal may lead to myxœdema. Sarcoma of the thyroid is very rare; it is usually a round-celled rapidly growing tumour.

### Granulomata and Cysts

Miliary tuberculosis may affect the thyroid, and in secondary syphilis the gland may enlarge or a gumma may form later. A hydatid cyst may also develop in the gland. The cystic adenoma has been described on p. 648. A degeneration cyst is due to degeneration of an adenoma. Hæmorrhage may occur into it. Retention cysts are also described.

## THE PARATHYROID GLANDS

**Introductory.** There are usually four parathyroid glands, situated in close relationship to the middle third and lower poles of the posterior surface of the lateral lobes of the thyroid gland. Small accessory glands may be present in the upper or lower pole of the thymus or in

the carotid sheath. They are thought to be concerned with maintenance of calcium and possibly of phosphate balance in the blood. An extract (parathormone of Collip) on intramuscular injection causes a rise in blood calcium and increased output of calcium in the urine. The calcium is derived from the bones. Deficiency of vitamin D results in lack of calcium in bone, and excess may cause hypercalcaemia. Probably vitamin D regulates calcium absorption and excretion in the intestines, but it is not evident that it affects parathormone activity. Parathyroidectomy in mammals causes tetany as the result of the fall in the blood calcium.

### Hyperparathyroidism

*Acute hyperparathyroidism* is rare. The symptoms resemble those resulting from the injection of parathormone into the dog. It may result from the accidental over administration of parathormone, or be due to a parathyroid adenoma. The chief features are anorexia, nausea, vomiting, drowsiness, constipation, and low blood pressure. The blood urea and calcium are raised. The condition is liable to be mistaken for uraemia. *Chronic hyperparathyroidism* results in generalised osteitis fibrosa (see p. 605).

### Hypoparathyroidism

Deficient activity of the parathyroids results in tetany. The relationship of tetany to the parathyroid function is not clear in all cases.

### Tetany

#### (*Spasmophilia*)

**Definition.** A symptom complex characterised by over excitability of portions of the nervous system, with resultant muscular spasm.

**Etiology.** There are two main causes—

1. **Deficiency of calcium in the blood.** In many cases of tetany the blood calcium is low. Injection of parathormone or the administration of calcium by mouth, intramuscularly or intravenously, raises the blood calcium and relieves the spasms of tetany. Calcium occurs in the blood in three forms, 50% is in organic combination, and 50% is present either in un-ionised inorganic form or as calcium ions. Tetany may be due to a deficiency of calcium ions. In some cases the blood calcium figure is low, in others the ionised calcium may be low although the total serum calcium is normal. 2. **Alkalosis.** This does not result in a decrease in the serum calcium, but it may lower the amount of ionised calcium. This has not been proved, and there is experimental evidence against it. Other explanations are that the alkalosis increases the excitability of nerve fibres or nerve centres, or limits the available supply of oxygen to the muscles by altering the dissociation curve of oxyhaemoglobin.

Tetany may result from 1. **Parathyroid Deficiency.** The blood calcium is low. It may result from operations on the thyroid gland.

(tetania parathyreopriva). It also occurs in Graves' disease where the parathyroids may be involved, and in epidemics possibly due to parathyroiditis. 2. *Deficient Absorption of Calcium*. Infantile tetany associated with rickets results from deficient absorption of calcium owing to lack of vitamin D. Osteomalacia in adults may be due to a similar cause. Tetany may also complicate cœliac disease, sprue, dysentery, cholera and Hirschsprung's disease. 3. *Alkalosis*. Gastric causes include pyloric stenosis and the administration of large doses of certain alkalis in the treatment of peptic ulcer. It has been noted in chronic arsenic poisoning, in which there is prolonged vomiting. It may also occur in high intestinal obstruction and nephritis. Hyperpnœa is a comparatively common cause of tetany. It may be hysterical, voluntary, or result from excessive exercise. The serum phosphorus is found to be low if the blood is taken immediately after an attack of hyperventilation tetany. In very hot and humid climates hyperpnœic tetany may occur, and fireman's cramp in some cases is probably due to alkalosis, in others to loss of chlorides.

Tetany is said also to occur as the result of certain poisons such as chloroform, lead and morphine, and to be associated with some nervous diseases such as syringomyelia or cerebral tumours. The explanation of these conditions is obscure, unless there is hyperventilation. It may also be associated with pregnancy and lactation, possibly due to excessive demands for calcium, and to deficient absorption of calcium as in osteomalacia.

**Pathology.** Lesions may be found in the parathyroids, such as inflammation, hæmorrhage, fibrosis, miliary tubercles, leukæmic infiltration, and a tumour, or there may be no lesion present. The spasms affect chiefly small voluntary muscles; it is not known whether they are neurogenous or myogenous in origin, but certainly both motor and sensory nerves are hyperexcitable. It is doubtful if involuntary muscles are affected. Cataract may be due to deposition of calcium phosphate in the lens, and calcification of cerebral arteries may be a cause of convulsions.

**Clinical Findings.** Infantile tetany occurs in rickety children between the ages of 6 months and 2 years; they often have diarrhœa. The tetany may show itself by attacks of laryngo-spasm (*Laryngismus stridulus*, see p. 119) or by carpo-pedal spasm (see below), or by general convulsions. A case of post-operative tetany of mild degree illustrates well the condition seen in adults. The patient complains of attacks of tingling or numbness in the fingers, with stiffness in the fingers or arms, and then contractions of the hands, the thumb being pressed across the palm causing pain. The feet may also be affected, becoming stiff, the ankles turning in and the toes being flexed. Twitching may be noticed in various muscles of the body, and the face and the lips may feel tight during an attack, causing difficulty in speech or swallowing. The attacks may come on suddenly, and be provoked by raising the arms or lying still in a fixed position. The patient may also have convulsive fits with or without loss of consciousness. Vision may periodically become dim, and finally be lost owing to cataract.

*On Examination.* During an attack carpo pedal spasm is typical. The hands are fixed in the accoucheur's position, with the thumb flexed across the concave palm, fingers adducted and flexed at the metacarpo phalangeal joint, and extended at the interphalangeal joints. The wrists and elbows may be flexed. The feet. The ankles are dorsiflexed and may be inverted, with toes flexed and the sole concave. The knees are extended. The feet and hands may be congested. The corners of the mouth may be depressed and the lips protruded (carp mouth). Spasm may also affect the abdominal muscles or diaphragm and intercostal muscles, causing expiratory apnoea, and lateral deviation of the eyes may occur. In a long standing case the skin is dry, the nails brittle, the teeth become carious and the hair falls out. Cataract may develop, and in its early stages can be detected by seeing opacities with the slit lamp. In severe cases general convulsions may occur in adults. The increased irritability of the neuro muscular system in latent tetany may be demonstrated by the following signs. *Chvostek's sign.* A tap over the facial nerve in front of the lobe of the ear provokes varying degrees of contraction of the muscles supplied, such as twitching of the angle of the mouth, of the outer canthus of the eye, cheek, forehead and alae nasi. *Trousseau's sign.* Compression of the upper arm by a sphygmomanometer armlet, with a pressure sufficient to obliterate the radial pulse, provokes spasm of the hand in 1 to 5 minutes. This may not be present in a well developed case. *Schultze's sign.* A localised dimpling occurs on tapping the protruded tongue with a patella hammer. *Pool's sign.* Extension of the brachial plexus, by forcible abduction of the arm, causes spasm in the hand and arm. *Schlesinger's sign.* Flexion of the hip with the knee extended causes spasm of the leg. *Erb's sign.* There is increased excitability of motor nerves to galvanic stimulation. A kathodal opening contraction will occur with a current of less than 5 milliamperes, applied to such a nerve as the external peroneal. *Hoffman's sign.* Stimulation (mechanical or electrical) of a sensory nerve will cause a muscular spasm. In post operative tetany the serum calcium is usually below 7 mg per 100 c.c. As the calcium falls the serum phosphorus rises. In hyperventilation tetany the blood calcium is normal, but the phosphorus is low.

*Diagnostic* In infants other causes of convulsions must be considered (see p. 293). Laryngospasm must be differentiated from other causes of laryngeal obstruction (see p. 119). Carpo-pedal spasm occurs only in tetany and is diagnostic. In adults, generalised convulsions due to tetany have been mistaken for epilepsy, the patient does not, however, always lose consciousness, and there is much pain. Tetanus and strychnine poisoning are usually easily differentiated. Between the spasms the special signs described above are of value. Hysterical spasms are not usually bilateral.

*Course and Complications.* Recurrence is very liable to occur, the attacks may last a few minutes, or persist for hours or days. Cataract is an important complication.

*Prognosis.* Tetany is a serious condition. In infants it may prove fatal with laryngospasm or generalised convulsions, and in adults it is

especially serious in association with childbirth, gastric dilatation or nephritis, or after operation on the thyroid.

**Treatment. Prophylactic.** In infants prophylactic measures are to be directed to the prevention of rickets (see p. 621). Tetany is rare after goitre operations in England, but commoner abroad where more radical operations are performed.

**Curative.** The spasms, if violent, may be controlled by administration of chloroform as a temporary measure of urgency. Morphine should never be given. If the blood calcium is below normal, a diet rich in calcium, such as eggs, milk and green vegetables, should be given. The optimum daily intake of calcium for a child is 1 G., and for an adult 0.75 G. A quart of milk contains 1 G. of calcium. Calcium should also be administered as calcium lactate gr. 5 to gr. 240 t.d.s. by mouth, or injected intravenously as calcium gluconate 10 mls daily, until the symptoms are relieved. When calcium is given by mouth vitamin D should also be given, such as liq. calciferol. (B.P. Add.) (see p. 621) daily. High potency Ostelin tablets containing 50,000 i.u. may be given, 2 tabs. daily for [5 days a week, without additional calcium. Even when the blood calcium is below 6 mg. per 100 c.c., it is seldom necessary to give intramuscular injections of parathormone. A fraction of irradiated ergosterol, dihydrotachysterol or A.T.10, which does not contain vitamin D, when taken by mouth raises the blood calcium. Five to 10 mls are taken daily, but blood calcium estimations are essential as hypercalcaemia is liable to occur. Parathyroid extract by mouth has no effect in raising the blood calcium, as the active principle is destroyed in the stomach. Cataract requires treatment by operation.

Constipation should be relieved by an enema, and a colonic washout given every other day, with 1 to 2 pints of normal saline, until the muscular excitability is normal. Sedatives such as chloral hydrate gr. 5 to 10 b.d.s. by mouth for an adult may be necessary for the first 2 or 3 days. If there is gastric dilatation, a tube should be passed and gastric lavage performed with normal saline. When the calcium in the blood has been restored to normal the patient should be kept on a diet rich in milk and eggs, and calcium lactate, gr. 5 to 10 given daily. Spontaneous hyperventilation tetany can be terminated by breathing air containing excess of  $\text{CO}_2$ . Tissue acidosis resulting from muscular activity may be the cause of spontaneous termination of hyperventilation tetany. In other cases of tetany due to alkalosis the treatment is as described on p. 88.

## THE SUPRARENAL GLANDS

**Introductory.** The suprarenal gland is composed of two parts, a cortex and medulla, which in elasmobranch fishes remain separate; the cortex forming an inter-renal body. Further, the carotid body, the paranglionic bodies and accessory suprarenals belong to the same system. The cortex is derived from mesoderm and is composed of cells containing lipid granules. The medulla is of sympathetic nervous origin and

contains chromophil cells around which preganglionic branches of the splanchnic nerves end. The cortex of the glands is necessary to life as it contains the active principle, which is a lipoidal substance and is available as Eucortone, Cortin or Eschatin. Vitamin C is also stored in the cortex. Swingle and Pfiffner have maintained life in suprarenalectomised rats by daily injections of a cortical extract, the dose being 1 ml per kg body weight. Desoxycorticosterone was subsequently synthesised. It is cheaper than Eucortone and has a very similar action. The internal secretion of the medulla is adrenaline, and it is inert on oral administration. The function of the cortex is uncertain, it is concerned with growth, sexual development and maintenance of life. Eucortone is concerned with the metabolism of sodium, chlorine and phosphorus, and with water distribution in the body. It appears to act on the kidneys for when it is lacking the amount of sodium and chloride falls in the blood and rises in the urine. Adrenaline acts on the junctional tissue between the sympathetic nerve terminations and the cells they activate. The results produced by adrenaline injection are similar to those caused by sympathetic stimulation. Adrenaline is poured forth into the blood in times of stress. The effects produced include dilatation of the pupils, rise of blood pressure, acceleration of the heart, erection of hairs, dilatation of bronchioles, transference of glycogen from muscles to the liver, increase in blood sugar and blood lactic acid, relaxation of intestinal muscles and constriction of the intestinal sphincters and of the arterioles of the skin and splanchnic region.

### Hyperadrenia

Hypertrophy and tumours may result in overactivity of the suprarenals.

*The Cortical Tumours* These include an adenoma, adenocarcinoma and carcinoma. An adrenal carcinoma often involves the kidney and metastases occur in abdominal and mediastinal glands and in the lungs, liver and brain. The tumour may be functionally inert causing pressure effects or physiologically active resulting in virilism or feminism. With the physiologically active tumours diagnosis is possible before metastases have occurred and removal of the tumour is often successful. The condition is known as hypercortico adrenalism or the adrenogenital syndrome.

*The Adrenogenital Syndrome* This may affect children or adults, due to a simple or slowly growing malignant tumour. In boys the symptoms include excessive muscular development, a low voice and premature sexual development (the infant Hercules). Girls assume male characteristics. The onset of menstruation is delayed, the skin of the face is coarse and red, the voice harsh, the pubic hair increases, there is obesity and the clitoris may enlarge. In adults the symptoms and signs closely resemble those of Cushing's syndrome (see p. 668). There is a similar distribution of obesity, facial hirsuties, loss of sexual desire, and in women amenorrhœa. There may be a persistent glycosuria and high blood pressure. A direct X-ray examination of the renal region and one taken after the injection of Uroselectan

(iodoxylum B.P.Add.) may suggest the presence of a suprarenal tumour. The diagnosis is aided by examination of the urine for the presence of the male sex hormone. The normal secretion of androgens is 5 to 15 mg. "sterone" a day. Female patients with a cortical tumour often excrete over 100 mg. "sterone," and with male patients a moderately high androgen excretion is found. The Ponceau fuchsin stain with a counter stain of aniline blue is of value in confirming the diagnosis when the suprarenal has been removed and can be examined microscopically. The normal suprarenal cortical cells stain blue, in virilism they stain red. Removal of the tumour may effect a cure.

*The Medullary Tumours.* A paraganglioma may result in paroxysms of high blood pressure, with nausea, vomiting, shivering, sweating, pallor, tachycardia and fever. Other medullary tumours include the ganglio-neuroma, which is benign, and the neuro-blastoma which is very malignant. The latter affects children. When the right suprarenal is involved metastases occur in the liver (Pepper syndrome), and with the left suprarenal the bones and left eye may be affected (Hutchison syndrome).

In Graves' disease (see p. 649) or after shock, overactivity of the suprarenals may be present with diminished sugar tolerance and glycosuria, the sugar being derived from glycogen in the liver and muscles, and possibly from protein. At the climacteric some of the symptoms such as rise of blood pressure may be due to hyperadrenia.

### . Hypoadrenia

The most important manifestation of hypoadrenia is Addison's disease. Other conditions which may be associated with hypoadrenia are :—Circulatory failure and shock symptoms as in diphtheria, typhoid fever, scarlet fever and cholera. Haemorrhagic necrosis of the suprarenals may be the lesion in these cases. Acute suprarenal haemorrhage involving the medulla may occur in the fulminating type of meningococcal meningitis, associated with a petechial rash. This is usually rapidly fatal. Neurasthenia may result from a functional disturbance of the suprarenal glands. In congenital hydrocephalus the medulla of the suprarenals has been found absent in some cases. Progeria may be due to a hypoplasia of the cortex.

### Addison's Disease

**Definition.** A disease characterised by low blood pressure, gastrointestinal disturbances, weakness and pigmentation of the skin.

**Etiology.** Addison's disease is caused by a lesion of the suprarenals, and less commonly of the splanchnic ganglia. It is rather more frequent in males.

**Pathology.** Addison, in 1855, based his description on 11 cases; of these the suprarenals were tuberculous in 5, carcinomatous in 3, fibrotic in 2, and an acute haemorrhage had occurred in 1. In 4 of his cases only 1 gland was affected. The glands may also be gummatous, atrophic or suppurative, or they may be normal, but the splanchnic ganglia are then diseased. The low blood pressure is probably due to

loss of sodium The gastro-intestinal disturbances may be due to unbalanced vagal activity or to disturbances of water metabolism, alkalosis, and a rise of blood urea, the general weakness to unneutralised toxins and to a deficient sugar supply to the muscles, and the pigmentation to a disturbance of metabolism whereby tyrosine, normally present in the skin, is changed by tyrosinase into melanin instead of being converted into adrenaline The excess of melanin may be due to a deficiency of ascorbic acid (see p 618), which is normally stored in the suprarenal cortex and which inhibits pigment formation The localised distribution of the pigmentation suggests that cutaneous vaso dilatation plays a part

**Clinical Findings** The patient is usually an adult between the ages of 20 and 40 who notices progressive weakness, loss of appetite, wasting, darkening of the skin, and coldness of the extremities Pigmentation may precede the other symptoms of Addison's disease by several years Vomiting may occur from time to time or be very persistent, and there may be diarrhoea Rarely the disease has an acute onset

**On Examination** The patient appears somewhat wasted There is chestnut brown pigmentation of the skin, especially on the face, the backs of the hands axillæ, round the nipples and navel, in the pubic region, and where there is pressure as at the waist or garter line Small black spots may be seen, especially on the forearms, leucodermic patches may also occur, and the hairs growing from them may be blanched Slaty pigmentation may occur on the tongue, the buccal mucous membrane, or in the conjunctivæ, anus or vagina The blood pressure is low, usually below 100 mm Hg systolic The pulse is somewhat frequent and the temperature usually subnormal, apart from active tuberculosis Calcification of the suprarenals may be seen in some cases on X ray examination of the abdomen The blood Anæmia may be present, or the blood may be viscid There may be a relative lymphocytosis with an eosinophilia up to 6% The blood urea and non protein nitrogen rise in the penultimate stage There is a marked fall of the blood sodium and to a lesser degree of the chloride and a rise of the potassium The urine Sp gr 1,008 to 1,012 A trace of albumin and a few red cells are usually present The basal metabolic rate is low

**Differential Diagnosis** The weakness and pigmentation may suggest pernicious anæmia, this is excluded by a blood count Other causes of pigmentation and gastric disturbance must be considered, such as arsenic poisoning, abdominal growths and pregnancy. Carcinoma of the stomach is excluded by the fractional test meal, opaque meal, occult blood test in faeces, and the absence of a tumour or of secondary deposits It may, however, co-exist with Addison's disease In suspected arsenical poisoning the hair should be tested for arsenic Other causes of pigmentation which may have to be considered are race, sunburn, dirt, silver and lead poisoning, hæmochromatosis, abdominal tuberculosis, and Graves' disease A systolic blood pressure much over 100 mm Hg renders the diagnosis of Addison's disease very unlikely The Cutler sodium chloride deprivation test is of value in



the diagnosis of doubtful cases of pigmentation, and in vomiting associated with pulmonary tuberculosis in order to determine whether the suprarenals are affected. The patient is given a low sodium and high potassium diet, and the urinary sodium or chloride is estimated during the last 4 hours of a 52 hours' period. In Addison's disease, although the blood sodium and chloride are low, the urinary sodium chloride may be as much as six times greater than normal. Under the conditions of the test an urinary chloride in excess of 225 mg. per 100 c.c. is indicative of adreno-cortico insufficiency. This test is dangerous and unnecessary, as the diagnosis is obvious, in severe cases of Addison's disease, for fatal coma may be precipitated. For details of the test see Cutler, Power and Wilder, *J.A.M.A.*, 1938, 111, 117.

**Course and Complications.** The course is usually progressive, and complications such as intercurrent infections or myocardial degeneration may ensue. Crises may occur in which the patient is collapsed and appears moribund. Diabetes mellitus is a very rare complication.

**Prognosis.** This is very grave, death may occur in a few weeks or be delayed for several years. Recovery has been recorded in some instances. The most favourable cases are those in which the suprarenal lesion is simple atrophy. Active pulmonary tuberculosis is a very grave complication. The prognosis has undoubtedly been improved by the introduction of the special treatment detailed below.

**Treatment.** The principles of treatment of a subacute or chronic case consist in the administration of cortical extract or a synthetic preparation together with sodium chloride and water, and restriction of the potassium intake. Sodium is life and potassium is death to the sufferer from Addison's disease. Eucortone may be given intravenously, intramuscularly or subcutaneously in doses of 10 to 20 mls daily for a moderate case, the maintenance dose being worked out subsequently, and this may be 10 mls once or twice a week. A synthetic preparation, desoxycorticosterone acetate, dissolved in sesame oil, such as D.O.C.A., Percorten, or Syncortyl, may be preferred in doses of mg. 5 to 10 daily injected intramuscularly, and later mg. 5 once or twice a week. This is cheaper than the cortical extract, but in some cases relapses occur when the synthetic preparation is used, possibly due to the lack of some factor present in the natural extract. Pellets of the synthetic preparation may be implanted subcutaneously to obviate the administration by injection. The effect of four mg. 50 pellets thus implanted will last for 2 to 3 months. Sodium chloride should be administered in doses of 5 G. t.i.d. given in capsules, milk, or water flavoured with syrup of lemon.

The patient is often dehydrated and plenty of water should be taken. Excessive doses of sodium chloride and water may eventually lead to hypertension, œdema and congestive heart failure. The patient should be examined every month to prevent such complications developing. The diet should contain less than 1.5 G. potassium daily. A normal diet contains about 4 G. potassium. Substances rich in potassium which should be avoided include peas, beans, lentils, soups, dried fruits and chocolate.

Should a crisis occur, the patient should be kept warm and the bed heated with an electric cradle. An intravenous drip should be given using a solution containing dextrose 100 G, sod chlorid 10 G, sod citrat 5 G, Eucortone 50 mls, and water to 1,000 mls. Up to 150 mls Eucortone may be required in the 24 hours.

Operation may sometimes be required for the removal of a tuberculous suprarenal gland and kidney. The patient should be stabilised as described above, and on the morning of the operation an intravenous injection should be given of 2 litres of the above solution with Eucortone 20 mls per litre. After the operation this should be repeated once or twice.

### THE PITUITARY BODY

**Introductory** The pituitary body is essential to life. It is composed of the following parts. *The anterior lobe* (pars distalis) formed of chromophobe or agranular, and chromophil or granular cells in columns, the latter being eosinophil or  $\alpha$  cells and basophil or  $\beta$  cells, with connective tissue and blood vessels. It is thought that the chromophobe cells do not produce an internal secretion, but a secretion is derived from the chromophil cells. The anterior lobe produces many hormones, the most important of which are the growth hormone, the gonadotropic, the thyrotropic, the adrenotropic, the pancreatotropic, the diabetogenic and ketogenic, the parathyrotropic, and the lactation (prolactin) hormones.

The growth hormone is thought to be produced by the eosinophil cells. The gonadotropic hormones are believed to influence both the female and male genital systems. One hormone, Prolan A, known also as the follicle stimulating hormone (FSH) in the female causes growth of the ovarian follicles and the liberation of the oestrogenic hormone, oestradiol (see p 675). In the male it acts on the testis and is concerned with spermatogenesis. The other hormone, Prolan B, or the luteinizing hormone (LH), is responsible for the formation of the corpus luteum and the secretion of the ovarian hormone, progesterone (see p 675). In the male it is concerned with the descent of the testicle and provokes the internal secretion of the interstitial cells, which is known as testosterone (see p 675). Oestradiol is excreted in the urine both of women and men, and in women the amount excreted increases during pregnancy and during certain phases of the menstrual cycle. Progesterone is excreted in the urine as pregnanediol, and testosterone is excreted as androsterone. The presence in the urine of an anterior pituitary like luteinizing gonadotropic substance, originating in the chorionic tissue of the placenta, forms the basis of the Aschheim Zondek test for pregnancy. It produces haemorrhagic follicles in the ovary of the sexually immature mouse. A similar substance is found in the urine in chorion epithelioma, hydatidiform mole, and malignant disease of the testis.

**Clinical Applications of the Gonadotropic Hormones** The available preparations are not usually derived from the pituitary but from the serum of pregnant mares and from pregnancy urine. They are pre-

sumably formed in the placenta. Those obtained from pregnant mares' serum more completely resemble the two pituitary gonadotropic hormones than do those derived from human pregnancy urine, which are chiefly luteinizing in character. The former include preparations such as Antostab, Gestyl, Gonandyl, and Serogan, and those from pregnancy urine include Antuitrin S, Gonan, Physostab, Pregnyl, and Prolan. They are used for the treatment of pituitary infantilism, undescended testis and sexual under-development, and for certain types of metrorrhagia and sterility.

*The Pars Tuberalis* is difficult to demonstrate in man; its function is not known.

*The Pars Intermedia.* This is very variable in amount. It surrounds the pars nervosa and contains basophil cells. It secretes intermedin which has a melanophore-expanding effect on frogs and may also possess antidiuretic properties.

*The Pars Nervosa* consists of neuroglial cells, ependymal cells and pyramidal granular cells, called pituicytes. There are also present non-myelinated nerve fibres, hyaline masses and blood vessels. Pituiratin is secreted, probably from the pituicytes. This has been subdivided into oxytocin (Pitocin) stimulating the uterus and vasopressin (Pitressin) which is antidiuretic, raises the blood pressure, stimulates intestinal and other involuntary muscle and possibly raises the blood sugar by its effect on the glycogen in the liver, and is concerned with fat metabolism in the liver, preventing obesity. The secretion enters the blood stream and the posterior lobe secretions are not found in the cerebrospinal fluid.

The anterior lobe, pars tuberalis and pars intermedia constitute the glandular division and are derived from Rathke's pouch of the stomodæum, and the pars nervosa, infundibular stem, and median eminence constitute the neural division and are derived from the floor of the fourth ventricle. The posterior lobe is composed of the pars intermedia and the pars nervosa. Disturbance of function is known as dyspituitarism, which may be in the sense of overactivity of any part or parts of the gland, constituting hyperpituitarism, or underactivity known as hypopituitarism.

### Hyperpituitarism

Overactivity of the pituitary may give rise to sexual precocity or gigantism if it occurs before growth has ceased, or to acromegaly if it begins later.

In infancy hyperpituitarism may cause hemihypertrophy of the whole of one side of the child, or one leg only may be affected. This usually becomes unnoticeable as the child grows.

During childhood gigantism (height over 79 inches) may ensue or pituitary glycosuria. Thus a boy of 9 years of age may be over 6 feet tall.

In adult life acromegaly may develop, or virilism in women associated with a basophil adenoma of the anterior lobe. *Cushing's syndrome* is associated with such a tumour, which may be very minute.

Young people are chiefly affected. Males are usually tall and females short. There is adiposity of the face, neck and trunk but not of the limbs. Purplish cutaneous striae are seen on the lower part of the abdomen and on the thighs. The blood pressure is raised and there is hypertrichosis in women of the chin, upper lip and side whisker areas. The pubic hair has a male distribution. Hair tends to fall out on the head. Erythrocytosis occurs in some cases. The bones may be soft. In some cases there is glycosuria. There is usually impotence or amenorrhoea. In addition to the pituitary lesion the testes or ovaries may be found atrophied post mortem. The adrenals are often enlarged and may contain secondary adenomata. Cushing's syndrome, when occurring in women is difficult to diagnose from the adreno genital syndrome. The special tests described on p 603 should be carried out, namely estimation of androgen excretion in the urine and X ray examination of the kidneys before and after Uroselectan (iodoxylum B.P. Add.) injection. X ray treatment to the skull may afford relief. A pituitary tumour (usually an acinar adenoma) may cause pressure symptoms such as loss of vision and headache, without symptoms of secretory disturbance. If it presses on the basophilic tissue, sexual deficiency symptoms such as amenorrhoea may occur. There may be optic atrophy with leg pains and absent knee jerks (pituitary tabes) differing from tabes dorsalis clinically in that the pupils react to light.

Stalk tumours (suprasellar cysts) cause headache and loss of vision. The sella turcica is often distorted and a shadow may be seen above it by X rays. There may be early optic neuritis and later atrophy, clinically it closely resembles an adenoma of the pituitary. A suprasellar endothelioma may also occur.

*Pituitary Glycosuria* The mechanism of its production is obscure but the anterior lobe is thought to produce a diabetogenic hormone which may cause glycosuria possibly by damage to the  $\beta$  cells of the pancreas. A glycotrophic substance is also thought to be secreted which inhibits the action of insulin and so raises the blood sugar. Pituitrin also inhibits the action of insulin. A ketogenic hormone is also believed to be formed in the anterior lobe and it may be distinct from the diabetogenic hormone. The pancreatotrophic factor is thought to increase the production of insulin possibly by stimulating the growth of the  $\beta$  cells. Its effect therefore will be to lower the blood sugar. Clinically pituitary glycosuria may be associated with a pituitary tumour or with basal meningitis.

### Acromegaly

**Definition** A disease characterised by enlargement of the extremities.

**Etiology** Acromegaly is caused by oversecretion of the pituitary (chiefly anterior lobe).

**Pathology** There is usually an eosinophil adenoma of the anterior lobe, less frequently the tumour is a glioma, endothelioma or sarcoma. There is often hyperplasia of the adrenal cortex.

**Clinical Findings.** The patient is usually between the ages of 20 and 40 at the onset. The disease may first show itself directly after pregnancy. The sex incidence is practically equal. The patient may first notice tingling or numbness of the hands and feet, and later enlargement of the head, face, hands or feet. He then complains of headache, often bi-temporal, and of visual disturbances such as dimness of the outer part of the fields of vision. Amenorrhœa may be the first symptom. Early there may be sexual excitement, but mental torpor develops later. There may also be loss of smell, trigeminal neuralgia or nasal discharges of blood, mucus or cerebro-spinal fluid. In some cases the patient has attacks, known as "uncinate fits" with an aura of smell or taste, and convulsions may follow.

**On Examination:** In an established case the appearance is characteristic; the lower jaw, the malar bones and orbital ridges are prominent and the skull is enlarged. Twenty per cent. of patients are over 5 feet 11 inches in height. The tongue is big, the teeth spaced, the nose broad, the skin rough, thick, and it may be dark. The hands and feet are enlarged, the chest big, and there is frequently kyphosis. Pierre Marie described two types of hand, the *type en long*, and the *type massive*, which is spade-like (see Fig. 65, facing p. 651). The thyroid gland is often palpable. The temperature is low in the later stages of the disease. The blood pressure may be raised.

**Special Examinations.** The eyes: The characteristic change is bi-temporal hemianopia, due to pressure of the tumour on the decussating fibres of the optic nerve in the chiasma. This is determined by plotting the fields of vision. Early changes include primary optic atrophy and a scotoma for red. Optic neuritis rarely results from generalised increased intracranial pressure. The third, fourth or sixth cranial nerves may be compressed in the cavernous sinus with weakness of the ocular muscles. A stereoscopic X-ray of the skull: This may show enlargement of the sella turcica or erosion of one or both of the anterior or posterior clinoid processes. X-ray of the hands: A "tufting" appearance may be seen in the terminal phalanges which show lines radiating like a fan. Sugar tolerance test: In the early stages the tolerance may be diminished, the blood sugar rising above the renal threshold and glycosuria resulting. Later there is hypopituitarism with increased sugar tolerance. Basal metabolic rate: This may be increased early and diminished later in the disease.

**Differential Diagnosis.** The appearances of a developed case are typical. In the early stages the special examinations mentioned above will help in the detection of a pituitary lesion.

**Course and Complications.** The course is usually progressive, but there may be intermissions. Certain types are described: 1. *Benign*. Duration up to 50 years. 2. *Chronic*. Duration 8 to 30 years. 3. *Acute malignant*. Duration 3 to 4 years. 4. *Cephalic*. Head only affected. 5. *Stationary*. 6. *Formes frustes*. The headaches may become excruciating and blindness or dementia occur. Death may result from heart failure, convulsions, diabetes mellitus, uræmia or intercurrent infections such as pneumonia and tuberculosis.

**Prognosis** This is very grave, but there is a tendency to arrest in some cases, and a cure has been effected by operation

**Treatment** X ray treatment to the skull may afford relief Operation is usually advised on account of threatened loss of vision or severe headache A decompression and incision of the capsule of dura may relieve headache, even if the tumour is not removable In the early stages thyroideum, increasing from gr 1 to gr 5 daily, may also be of value Insulin and dietetic restrictions may be necessary for the glycosuria In the later stages of hypopituitarism daily subcutaneous injections may be given of 1 mil of anterior lobe extract (Antuitrin)

### Hypopituitarism

The effects produced by under activity of the pituitary vary with the age of the patient. The characteristic features are sexual inactivity, infantilism deposition of fat, lowered metabolism, and oversecretion of urine The lesion, if an adenoma, is of the chromophobe type, and probably produces its effect by pressure on the chromophil cells of the pituitary In other cases the symptoms are due to a persistence of chromophobe cells and improvement occurs as more granular cells are formed

### Pituitary Infantilism

*The Loran Type (Ateliosis)* The child does not grow, the trunk especially being short The intelligence is good The sexual organs are small but there is no adiposity It is considered to be due to insufficiency of the anterior lobe

*Fröhlich's Type (Dystrophia adiposa genitalis)* This was originally described in a boy of 14 but may not commence until adult life There is adiposity affecting the arms legs and trunk, especially around the pelvis and below the scapulae The skin is smooth, the fingers tapering, the genital organs immature, the intelligence is usually normal but there is a tendency to somnolence (fat boy of Dickens) The blood pressure is often slightly raised The temperature is subnormal and the sugar tolerance is increased Males tend to conform to the female type This variety occurring in children is sometimes called the *Brissaud type* Another variation occurring in children, in which the face is round, is called the "pudding face" type of Fearnside They are thought to be due to insufficiency of both lobes of the pituitary The *Laurence Moon Birdl syndrome* is characterised by obesity, polydactylism mental deficiency, and retinal changes resembling those of retinitis pigmentosa

### Hypopituitarism in Adults

There is usually general obesity, especially around the pelvic girdle, and diminution of sexual function The male skeleton and pubic hair distribution tend to conform to the female type The fingers become delicate and tapering and the skin smooth In females there is usually amenorrhœa

*Simmond's Disease* (Hypophyseal cachexia. Splanchnomicria). This is due to deficiency of the anterior lobe, which is often found to be replaced by scar tissue, or which may be the site of an embolus, thrombosis or hæmorrhage. It occurs in adults usually over the age of 30, and affects both sexes. There is usually marked emaciation, with trophic changes in the teeth, hair and nails, loss of appetite, vomiting and profound weakness. Sexual power disappears, the temperature and B.M.R. are low. The similarity to anorexia nervosa has been referred to on p. 27. In some respects it resembles Addison's disease, but there is usually no pigmentation. Death usually occurs in 1 to 2 years. A case which was presumably due to a functional disturbance of the pituitary has been successfully treated by the injection every other day of 150 rat units of the anterior pituitary-like gonadotropic substance of pregnancy urine; 5,000 rat units were injected in 4½ months. In another case daily subcutaneous injections of anterior lobe extract (Antuitrin) 1 mil. daily, resulted in a gain of 15 lb. in 2 weeks.

*Dercum's Disease* (Adiposis dolorosa). This usually occurs in women after the climacteric. Painful, fatty, subcutaneous masses appear, especially in the upper arms and thighs, and there is general weakness and mental deterioration. In all cases of suspected hypopituitarism a sugar tolerance test should be performed. The tolerance is usually increased. The basal metabolic rate is low. An X-ray film of the skull may reveal alterations in the pituitary fossa, and visual disturbances may occur. The temperature is often about 97° F., and an injection of 1 mil. of the extract of the anterior lobe of the pituitary will cause a rise of temperature to 99° F. (Cushing's thermic test).

*Treatment.* This is not very satisfactory. If a tumour is present an operation may be necessary to relieve headache or preserve vision. In pituitary infantilism treatment should be begun with gr. ½ of thyroideum. Oral administration of pituitary extract is probably without effect. Children may be given subcutaneous injections of Antuitrin Growth, 2 to 5 mils twice a week. A gonadotropic factor obtained from pregnant mares' serum, such as Antostab, can also be injected, 1 mil. daily.

### Diabetes Insipidus

*Definition.* A disease characterised by thirst and persistent polyuria, the urine being of low specific gravity and containing no abnormal substance.

*Etiology.* Probably all cases are due to lack of the anti-diuretic pituitary hormone, associated with hypofunction of the posterior lobe, or with lesions of the hypothalamus near the tuber cinereum. The nucleus supraopticus in the hypothalamus is probably the controlling centre. Water-balance in the body is disturbed by loss of the oliguric hormone which normally affects the thin segment of the loop of Henle, stimulating it to absorb water and so to concentrate the urine. Normally 168.5 litres of glomerular filtrate are reabsorbed in 24 hours, and in diabetes insipidus this amount may be reduced to 130 litres. The secretion of the thyroid gland also appears to increase water elimination and

symptoms of diabetes insipidus are sometimes associated with thyrotoxicosis *Predisposing causes* 1 Heredity, especially in the benign idiopathic cases 2 Age 10 to 40 years 3 Sex Males predominate

*Pathology* The following lesions have occurred Sarcoma, secondary carcinoma, a chromophobe adenoma, gumma or leukæmic infiltration of the pituitary, injury to the sella turcica as by a bayonet or bullet wound or fractured base, basal syphilitic or tuberculous meningitis involving the inter peduncular space It is also a symptom of the Hand Schüller Christian disease (see p 512) and of post encephalitic Parkinsonism Experimental puncture of the hypothalamus, or compression of the infundibulum by a clip results in diabetes insipidus At autopsy there may be enlargement of the kidneys, ureters and bladder

*Clinical Findings* The patient is usually a young adult, who notices increasing thirst and the passage of large quantities of urine There may be a sudden onset after an injury He is usually constipated, the mouth dry, and sleep is disturbed by the desire for micturition Sometimes there is great hunger and thirst becomes unquenchable

*On Examination* In an established case there is usually emaciation with a dry skin Neighbourhood symptoms may be present if the lesion is a tumour The urine Specific gravity 1,001 to 1,005 Volume 10 to 40 litres daily There is no albumin but occasionally a trace of sugar may be present The blood The red cells may be over 6 millions per c mm The Wassermann reaction may be positive The sugar tolerance is normal or diminished The basal metabolic rate is usually normal An X ray film of the skull may show enlargement of the sella turcica

*Differential Diagnosis* Other varieties of polyuria and frequency must be excluded especially those due to chronic nephrosclerosis intermittent hydronephrosis diabetes mellitus, hysteria and an enlarged prostate The renal function tests and abnormal urinary constituents serve to diagnose chronic nephritis In hysteria and intermittent hydronephrosis the polyuria is inconstant, and a renal swelling may be felt in the latter Examinations of the urine and the blood for sugar differentiate diabetes mellitus

*Course and Complications* The hereditary type often runs a prolonged course for many years In other cases the course must vary with the cause. Complications include pulmonary tuberculosis and coma, the latter is the usual mode of termination

*Prognosis* This varies with the cause In idiopathic cases life may be little, if at all, curtailed Syphilitic cases may be cured by treatment In malignant cases the disease is usually fatal within a year

*Treatment* In syphilitic cases iodides and valerian should be given such as Pot iod gr 5 to 10, tnc valerian ammoniat m 30, sp ammon aromat m 20 aq menth pip dest ad fl oz 1 Fl oz 1 three times a day A salt poor diet may be given but the fluid intake should not be restricted Pituitrin has an antidiuretic effect, 1 mil may be required twice or three times a day intramuscularly or last thing at night, or 0.5 mil of Pitressin which is the active constituent If intestinal colic ensues 1 mil of Pituitrin (half strength) may be



as a nasal spray or on a little wool as a nasal plug as an alternative, but this is not always efficacious. Nasal insufflation of the powdered whole gland, mg. 25, 3 or 4 times daily and during the night, may also be tried. A more prolonged effect, lasting for about 44 hours, is obtained by the subcutaneous injection of 1 to 1.5 mil. of Pitressin Tannate (5 pressor units in 1 mil. of peanut oil). This treatment may have to be continued indefinitely. Thyroidectomy has proved successful in certain cases following encephalitis lethargica, and in those associated with thyrotoxicosis.

### THE THYMUS GLAND

The thymus increases normally in size until puberty, after which it gradually atrophies. It is probably concerned with growth, and a concentrate called thymo-crescin has been prepared from it, which on injection increases the general growth of rats.

Clinically, it is of importance in status lymphaticus and if a tumour arises in it. It is sometimes enlarged in exophthalmic goitre and in myasthenia gravis.

#### Status Lymphaticus

A condition characterised by enlargement of the thymus gland and lymphatic tissue generally. The symptoms include: Thymic asthma, due to pressure of the enlarged gland causing dyspnoea with stridor. This may be mistaken for croup due to laryngeal obstruction. Sudden death may occur during sleep or during the administration of an anæsthetic, possibly due to mechanical stimulation of the vagus. A status hypoplasticus. It is thought that some children with the status lymphaticus grow up, there being a compensatory overactivity of other glands such as the suprarenal or pituitary (Timme's multi-glandular syndrome). Enlargement of the thymus can be diagnosed by X-ray examination and treated by exposure to X-rays.

#### Thymic Tumours

These may be simple, such as a lipoma or fibroma; cystic, such as a dermoid; or malignant, such as a carcinoma or sarcoma. The tumour may also be due to Hodgkin's disease. The effects produced are mechanical, those of a superior mediastinal tumour.

Treatment. The tumour may be removed in some cases by an operation involving splitting of the sternum. If this is not possible, X-rays should be used.

### THE PINEAL BODY

The function of the pineal body is not definitely known. It probably forms no internal secretion but exerts through the nervous system a check upon sex development.

#### The Pineal Syndrome

A pineal tumour, such as a teratoma, may give rise to overgrowth and sexual precocity in boys but not in girls. It may also produce internal hydrocephalus with headache owing to obstruction of the

Sylvian aqueduct, pressure on the corpora quadrigemina or cerebellum may result in diplopia and fits, and may cause tremors of the extremities. One or other pupil may not react to light and external oculo motor paralysis may be present. X rays may reveal a pineal shadow. The prognosis is usually hopeless, as surgical removal of the tumour is rarely possible. X ray treatment may be given.

### THE TESTES

As described above (see p 667) the luteinizing hormone of the anterior pituitary is concerned with the descent of the testicle and provokes the internal secretion of the interstitial cells, which is known as testosterone. Testosterone can be synthesised and its propionate is available for clinical use. The treatment for failure of descent of the testicles, where there is no mechanical obstruction, consists in the injection of a preparation obtained from pregnancy urine such as Antuitrin S, 500 rat units (5 mls) twice a week for 3 months. Lack of development of the penis and testicles, or post pubertal eunuchism due to loss of the testicles by an accident, may be satisfactorily treated by the intramuscular injections of testosterone propionate, mg 5 to 25, daily. Methyl Testosterone may be given by mouth daily, mg 10 to 100 being usually required.

### THE OVARIES

*Ovarian Hormones* As described above (see p 667) the ovary, when stimulated by the pituitary gonadotropic hormones, secretes the oestrogenic hormone, oestradiol and the active principle of the corpus luteum, progesterone. Oestradiol is concerned with the growth of breast duct tissue, and with the proliferative changes in the uterine wall which occur during the first half of the menstrual cycle. Progesterone causes premenstrual changes in the uterine mucosa, is responsible for the implantation of the ovum and placental formation, and growth of the alveolar breast tissue during pregnancy. Oestradiol can be obtained from pregnant mares' urine, and oestradiol benzoate is used clinically as its absorption is slowed by benzylation. Synthetic oestrogenic analogues (such as stilboestrol and hexoestrol) are also now available for clinical use. Progesterone is obtained from the corpora lutea of sows or it can be synthesised.

*Clinical Applications* The value of these oestrogenic substances in the treatment of amenorrhoea is doubtful but primary amenorrhoea with infantilism has been treated by the injection of 250,000 international benzoate units of oestradiol spread over a period of 5 weeks. To inhibit lactation after childbirth stilboestrol, mg 5 to 10, given by mouth within 24 hours of the birth, is usually sufficient. Mg 1 to 5 t.i.d.s is required for the treatment of kraurosis and senile vaginitis, and for vulvo vaginitis in infants and children mg 1 tablet, crushed in milk, is given t.i.d. for 12 doses. Symptoms due to deficiency of ovarian secretion occur at the climacteric. They include amenorrhoea, headache, flushings, increase of weight and nervous instability. They usually pass

off in the course of 2 or 3 years. The nervous symptoms can be relieved to a certain extent by the administration of bromides such as sodium bromide gr. 5 to 10 t.i.d.s. The administration by mouth of stilbæstrol in doses varying from mg. 0.1 to 5 t.i.d. is very successful. The dose should be reduced as soon as the symptoms improve. Progesterone is used for the treatment of threatened abortion (mg. 5 to 10 daily for a short period), for habitual abortion (mg. 1 to 2 daily until the thirty-second week of pregnancy), and it is sometimes of value in menorrhagia (mg. 1 to 5 daily for 6 days).

## INFANTILISM AND DWARFISM

**Definition.** Infantilism is a condition characterised by delay of adult development with deficiency of secondary sexual characteristics. There may be no abnormality of growth. In dwarfism the secondary sexual characteristics, mental and bodily, are present, but growth is stunted.

### Infantilism

This may be due to: 1. *Deficiency of an internal secretion.* Thyroid deficiency as in cretinism (see p. 654), pituitary deficiency as in the Lorain type or Frohlich's type of infantilism (see p. 671). An infarct in the anterior lobe of the pituitary will also cause progeria. Suprarenal deficiency as in progeria (see p. 664), pancreatic deficiency as in pancreatic infantilism with inefficient fat digestion, renal deficiency as in renal infantilism associated with cardio-renal degeneration and dilatation of the bladder, ureters and pelvis of the kidneys (see p. 456), testicular and ovarian deficiency as in male and female eunuchism. 2. *Mongolism.* There is mental deficiency and the appearance of the child is characteristic. The palpebral opening slants downwards and inwards, the hands are broad, the index finger is short and the little finger curved with concavity to the radial side. In about 27% of cases two creases only instead of the normal three creases are present on the little finger of one or both hands, the distal and medial creases being replaced by a crease between the distal and proximal interphalangeal joints (Penrose). The foot "grasp-reflex" (see p. 311) is present in about 5% of cases. The tongue is often large, fissured and protrudes. The disposition is usually calm and affectionate. Enlargement of the thyroid gland and congenital heart disease may be present. The condition is incurable and not benefited by thyroid treatment. 3. *Congenital aberrations and diseases* such as microcephaly, congenital heart disease, anangioplastic infantilism (the arteries being improperly developed), persistent thymus and congenital syphilis. 4. *Acquired disease and infections* such as extensive bronchiectasis, ankylostomiasis, malaria and alcoholism.

### Dwarfism

This may be due to such conditions as rickets, renal dwarfism, spinal caries, osteogenesis imperfecta, achondroplasia, phocomelus, hydrocephalus and microcephaly.

## CHAPTER XII

### THE TROPICAL DISEASES

#### Malaria

**Definition** A disease characterised by recurrent attacks of fever, due to infection with a specific protozoon.

**Etiology** Malaria is due to infection with a protozoon, the *Plasmodium malariae*. Infection in man is caused by the bite of a female anopheline mosquito. The mosquitoes may fly normally 1 to 2 miles, or as far as 10 miles if aided by the wind. Three types of plasmodium affect man, the *P. vivax* causing benign tertian malaria, the *P. malariae* causing quartan malaria and the *P. falciparum* causing malignant tertian malaria. The incidence of malaria in a locality can be gauged by the spleen index, i.e., the proportion of the population with a palpable spleen, and the parasitic index as shown by blood slides. These run very closely parallel.

**The Life Cycles of the Parasite** The mosquito forms the definitive host and man the intermediate host. There are two cycles: an asexual cycle in man and a sexual cycle in a mosquito.

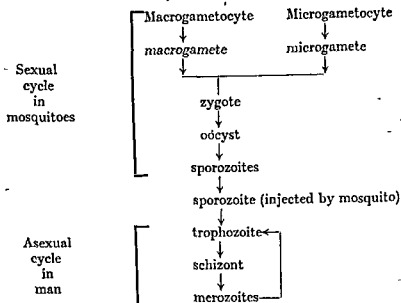
**The Asexual Cycle** The mosquito when it bites man introduces sporozoites. The sporozoite enters a red blood corpuscle and becomes a trophozoite. This grows and divides into segments forming a schizont (rosette), which ruptures, liberating merozoites into the blood. The merozoites may now enter other red cells forming trophozoites, thus completing the asexual cycle. The majority of the parasites are in the capillaries of the spleen and bone marrow and not in the peripheral circulation.

**The Sexual Cycle** Some of the trophozoites may enlarge and become macro- or microgametocytes. If these remain in the blood they die without further development. If, however, a mosquito bites a man when the gametocytes are present, they are taken into the mosquito's stomach, where further development occurs. The macrogametocyte becomes a female gamete and the microgametocyte forms a microgamete. A microgamete enters and fertilises a macrogamete and a zygote results. This passes through the mucous membrane of the mosquito's stomach and forms an oocyst. Numerous sporozoites develop in this cyst, which finally ruptures into the abdominal cavity, and the sporozoites pass to the salivary glands of the mosquito, and are injected into man by the mosquito's bite.

These stages can be represented diagrammatically as shown on p. 678.

The main distinguishing features between the types of parasite are as follows:

***Plasmodium Vivax*** This causes benign tertian malaria. The duration of the asexual cycle in man is 48 hours. The schizont is like a



rosette with about 18 merozoites. Schüffner's dots (deeply staining) are seen inside some of the red cells, which are enlarged and pale. The gametocytes are large and round.

*Plasmodium Malariae*. This causes quartan malaria. The duration of the asexual cycle in man is 72 hours, the schizont is like a daisy-head, with 6 to 12 merozoites. The red cells are normal. The gametocytes are large and round.

*Plasmodium Falciparum*. This causes æstivo-autumnal or malignant tertian or subtertian malaria. The duration of the asexual cycle in man is probably 48 hours. Schizonts are rarely seen in the peripheral blood, but they contain about 20 irregularly arranged merozoites. The gametocytes are crescentic, the female ones having more pointed ends than the male. *Predisposing causes*: 1. *Locality*: Malaria is rife in the tropics, especially in India, Africa and America. It also occurs in Southern Europe and is occasionally met with in South-east England. 2. *Temperature*: A mean temperature of 60° F. for at least 2 weeks is necessary for the development of the protozoon in the mosquito. Stagnant water favours breeding of the mosquitoes. 3. *Season and rainfall*: The effect of these upon the incidence of malaria varies in different localities. 4. *Age*: Children are especially susceptible, but all ages may be affected.

*Pathology*. The spleen becomes enlarged and very hard in chronic cases (ague cake). In acute cases it is soft and swollen. The liver may be enlarged and dark red. The brain may be dark slate-coloured. Parasites and pigment may be found in the vessels in various organs, such as the spleen, kidneys and brain.

*Clinical Findings*. The symptoms in the benign tertian and quartan types of malaria, apart from the time intervals of the fever, are usually indistinguishable. The patient complains of periodical attacks, beginning

generally about noon or later, of malaise, shivering, headache and perhaps nausea. He feels cold all over and may vomit. After a varying period of about 2 hours the skin becomes hot and flushed, and the headache is more intense. This phase persists for 2 or 3 hours, and is then followed by sweating and marked relief from the discomforts.

*On Examination:* During the initial cold stage the skin is blanched, but the body temperature rises to about  $102^{\circ}\text{F.}$  or  $104^{\circ}\text{F.}$ , and the

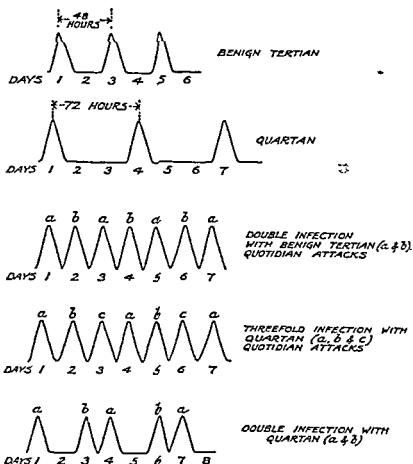


FIG. 66. DIAGRAM OF TEMPERATURE RECORDS IN MALARIA.

In double or threefold infections the attacks of fever may start at different hours, as (a) at noon, (b) at 8 p.m., and recur at the same hours on subsequent days.

pulse is frequent. The fever is thought to correspond with the liberation of the spores in the blood, perhaps due to freeing of their toxins, but there must be a threshold value, a definite amount of toxin being required in any individual to produce a rise of temperature. Thus patients may have schizonts in the peripheral blood without any fever, and again there may be fever without schizonts being found, although they are probably present deeper in the body. In the hot stage the temperature begins to fall, and in the sweating stage it reaches normal or subnormal. The whole attack lasts about 12 hours.

In the benign tertian infection the pyrexial attacks follow each other every third day; in the quartan every fourth day (regularly intermittent fever). Daily (quotidian) attacks may be due to a double infection with benign tertian, or to a threefold infection with quartan malaria, or in some cases to malignant infection. A mixed infection may cause irregular attacks of fever (see Fig. 66).

In malignant (subtertian) malaria attacks are liable to occur in the summer or autumn in temperate zones, but in the tropics this seasonal incidence does not prevail. In the less severe types the attacks may recur daily or every other day, or at irregular intervals. Frequently there are no rigors, but the fever may last for 24 hours or longer. Specially severe variations include (a) Cerebral malaria, in which the patient rapidly becomes unconscious with hyperpyrexia (temperature over 107° F.). In other instances there may be convulsions, paralysis or meningitic symptoms. (b) Algid type. The skin remains cold, and the body temperature may be subnormal or raised above the normal. There is great collapse and weakness. In some cases there is vomiting or diarrhoea, or hæmorrhages into the skin and various organs. (c) A bilious remittent fever. The characteristic features are the continuous temperature, jaundice and biliary vomiting. There is usually severe epigastric pain. In the typhoid type the patient is more ill, the tongue is dry and delirium present. Blackwater fever (see p. 682) is probably a variety of malignant malaria.

The spleen is usually palpable during the paroxysms, and in persistent cases remains so between the attacks, eventually becoming hard. It may reach to the umbilicus. The blood: When the temperature rises a film should be made, and stained for malarial parasites. Examination between the attacks usually yields negative results. In chronic cases a hæmolytic anæmia develops, and the large mononuclears may increase up to 20%. A positive Wassermann reaction may be obtained after the attack, but it is not universally accepted that this is due to the malaria. An indirect van den Bergh reaction is present in a large proportion of uncomplicated cases of subtertian malaria. The urine: Albumin is present in a few cases and hæmoglobinuria is the characteristic feature of blackwater fever.

**Differential Diagnosis.** In a malarial country malaria is the cause of the majority of feverish attacks, but when the patient has resided out of a malarial district for longer than three years the infection automatically dies out. Malaria must be diagnosed from other conditions, such as kala-azar, tuberculosis, amoebic hepatitis, typhoid fever, sunstroke, meningitis, cholera, dysentery, and pyæmic infections causing rigors. The diagnosis rests upon: (a) Finding the parasite in the blood. Prophylactic doses of quinine obscure the diagnosis, as then no parasites are likely to be present in the peripheral blood. (b) The response to quinine. If the temperature does not fall after adequate administration of quinine, the pyrexia is almost certainly not due to malaria. (c) The presence of a leucocytosis is also evidence against malaria.

**Course and Complications.** The course depends largely upon the treatment, the attacks in the benign tertian and quartan varieties are

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**Course and Complications.** The course depends largely upon the treatment, the attacks in the benign tertian and quartan varieties are



every case the urine and fæces should be disinfected as for typhoid fever (see p. 559).

### Yellow Fever

**Definition.** An acute disease characterised by fever, jaundice, albuminuria, and hæmatemesis.

**Etiology.** Yellow fever is probably caused by an ultra-microscopic filterable virus. In the urban and rural type of the disease the virus is conveyed from man to man, or from animals such as monkeys to man, by a mosquito, the *Aedes ægypti* (*Stegomyia fasciata*). Patients are infective to mosquitoes for the first 4 days of their illness. The virus undergoes changes in the mosquito so that the latter does not become capable of infecting man for 10 to 12 days, but remains infective for its lifetime (about 2 months). Indian monkeys can be infected by the blood of a patient, but West African monkeys are less susceptible. The serum of a patient who has recovered from yellow fever protects a susceptible monkey from the virus. Jungle yellow fever occurs in Brazil, but it is not known how man is infected, as the *Aedes ægypti* is not found there. It is probable that monkeys and other forest animals act as reservoirs of infection for urban and jungle yellow fever. *Predisposing causes*: 1. *Locality*: Yellow fever is endemic in West Africa and to a lesser degree in Brazil and Mexico. It was formerly very prevalent in Panama and Havana. 2. *Climate*: A mean average temperature of over 75° F. is necessary, and preferably a moist heat.

**Pathology.** The skin is very yellow. Hæmorrhages may be seen under the skin, in the muscles, stomach, intestines, pleuræ, meninges, kidneys and liver. The liver is soft and yellow, owing to fatty degeneration. The spleen appears normal. The kidneys show fatty changes, and casts may be found in the convoluted tubules.

**Incubation Period.** This is usually 3 to 6 days.

**Clinical Findings.** In the average severe type of the disease, the onset is sudden with a rigor and rise of temperature. The disease may fall into 3 stages. *The initial fever*: This lasts 3 or 4 days. The patient complains of severe headache, pains in the eyes, back and the calves of the legs. He may also have epigastric pain and vomiting, with constipation and insomnia.

*On Examination*: The face is flushed, the eyes bright, the skin dry, the pulse is somewhat rapid, about 120, and full. The urine diminishes and albumin appears in increasing quantities. Jaundice appears about the third day. The vomit at this stage usually contains bile, but some blood may be present.

*The period of remission*: This lasts 2 or 3 days. There is some improvement in the general condition of the patient. The temperature falls and the pulse becomes slow. The tongue is furred but the tip and edges are clean. Convalescence may now ensue, but usually the patient passes into the third stage of secondary fever. The temperature rises again, but the pulse rate remains slow (Faget's sign). The jaundice increases, the output of urine diminishes, much albumin is present with granular casts, red blood cells, hæmoglobin and bile. "Black vomit"

occurs, which is due to altered blood, and there may be diarrhoea with melæna. There are often hæmorrhages from the nose gums and into the skin. Death may follow or the patient may gradually recover.

**Varieties** 1 *Mild cases* There may be only slight fever with headache and a little vomiting, and the patient is well in a few days. 2 *Malignant cases* The temperature rapidly rises to over 106° F with severe vomiting of blood, suppression of urine, coma and death.

**Differential Diagnosis** The characteristic features of yellow fever are the jaundice, black vomit, massive albuminuria and slow pulse, with rising temperature. Mild cases may resemble dengue, but in the latter a rash is usually seen about the fifth day. Weil's disease may be hard to differentiate clinically, but it occurs in the absence of the mosquito which transmits yellow fever. The diagnosis of Weil's disease can be established by special laboratory tests (see p 698). In malaria the spleen is usually enlarged and parasites are found in the blood.

**Course and Complications** The course is as described above. Complications include Abscesses in the skin, parotitis and occasionally relapse.

**Prognosis** The average mortality varies between 20 and 60%. The outlook is unfavourable if the temperature rises to over 105° F or if there is suppression of urine.

**Treatment** *Prophylactic* The disease has been stamped out in Panama and other places by mosquito destruction. Water tanks should be protected by fine metal gauze covers and certain small fish may also be placed in the water to eat up the mosquito larvæ. Mosquito nets should be used at night. Mosquito control is not applicable for controlling jungle yellow fever. Prophylactic inoculation with attenuated yellow fever virus and immune yellow fever serum is now being practised with success especially in the jungle type of fever.

*Curative* The patient should be put to bed and kept warm. Barley water, alkalis and dextrose should be given by mouth in the form of dextrose orangeade or lemonade (8 oz of dextrose in 2 quarts of water, with the juice of 2 oranges or lemons) 3 to 4 pints daily and about gr 180 of sodium bicarbonate in the 24 hours sufficient to render the urine alkaline. The diet must not be increased during the period of remission except to give half-ounce feeds of half strength milk and lime water. During convalescence only easily digested foods such as eggs in milk soups etc should be given. Aperients should not be given by mouth after the onset of the illness. enemata should be used if required. The vomiting may be relieved by giving small drinks of iced champagne, or drop doses of liq iodi mitis m 2 in 1 oz of water every hour, and by applying a mustard leaf over the epigastrium. If the dextrose cannot be taken by mouth rectal salines containing 5% dextrose may be given in doses of 6 to 8 oz every 4 hours or an intravenous injection of 10 oz of normal saline containing 5% dextrose. If there is suppression of urine hot fomentations should be applied over the kidneys or the loins may be cupped. Cardio respiratory stimulants may be required for collapse symptoms such as Coramine (nikethamidum B.P. Add.) 1.5 mil or strychnin hydrochlor gr 1/60 hypodermically.

## Spirochætal Jaundice

(*Spirochaetosis Ictero-hæmorrhagica*. *Leptospiral Jaundice*. *Weil's Disease*)

**Definition.** A disease characterised typically by jaundice and hæmorrhages, due to a spirillum.

**Etiology.** Weil's disease is caused by the *Leptospira ictero-hæmorrhagica*, a coarse spirillum, whose dimensions are  $12\mu \times 0.25\mu$ . It is present in rats and is excreted in their urine, water thus becoming infected. The *L. canicola* is carried by dogs, and man may be infected. Transmission to man is probably through the skin, possibly by contaminated food, or by swallowing infected water while bathing in canals. *Predisposing causes:* Locality: Spirochætal jaundice occurs in Holland, Japan, Egypt, the Malay States, especially in mines, and was met with during the 1914-18 war in France and Gallipoli. Outbreaks have also occurred in Great Britain amongst sewer workers, tripe and fish cleaners, coal miners and farm workers.

**Pathology.** The body is usually jaundiced. Hæmorrhages may be seen in the mucous membrane of the stomach and duodenum, and in the lungs and kidneys. The liver may appear normal or fatty. The spleen is usually soft, and, in the types described by Weil, it is enlarged.

**Incubation Period.** This is probably about a week.

**Clinical Findings.** The onset is usually sudden and there may be a rigor. The patient complains of headache and severe pains in the limbs and back. There may be marked giddiness and vomiting or diarrhoea. Milder cases also occur.

**On Examination:** The conjunctivæ are injected, the temperature is high, about  $103^{\circ}\text{F}$ ., and the pulse comparatively slow, 80 to 90. Jaundice appears in about half the cases, beginning on the third or fourth day, when the skin itches. Petechial hæmorrhages may occur and bleeding from the gums or nose, and in some cases there is labial herpes which may be hæmorrhagic. Blood may be brought up from the stomach or lungs or passed in the motions. The temperature remains irregularly raised and falls by lysis, reaching normal about the eighteenth day. It may subsequently rise again for a few days. The tongue is dry. The spleen is not usually palpable but the liver may be felt. The glands in the axillæ and groins may be enlarged. The motions are usually constipated and pale. The blood: The leptospira is present for the first week of the illness and may be demonstrated either by blood culture or by intraperitoneal injection into the guinea-pig. A blood count shows a hæmolytic anæmia with a leucocytosis. The platelet count is low. The serum after the sixth day will agglutinate formalised cultures of the leptospira in a dilution up to 1/30,000. The adhesion test is also positive. In this test bacteria, such as the *B. coli*, adhere to the spirochætes in the presence of the patient's serum if he is suffering from the disease. The urine is scanty and contains albumin, bile and blood, and the leptospira is present after the tenth day. Jaundice does not occur in *L. canicola* infections.

**Differential Diagnosis.** The disease must be differentiated from jaundice due to enterica infections and from yellow fever. Other conditions such as catarrhal jaundice, and relapsing fever must be excluded. Cases in which there is no jaundice are liable to be mistaken for influenza. The diagnosis depends upon finding the leptospira in the blood or urine, on a positive agglutination or adhesion test, and the leucocytosis.

**Course and Complications.** Some cases pursue a severe course with intense jaundice and meningitis. Meningitis may occur apart from jaundice and the onset may be delayed for several months after the beginning of the disease. The diagnosis is established by finding the leptospira in the cerebro-spinal fluid and urine by guinea pig inoculation. Iritis may occur as a complication. Death may occur from cholæmia and uræmia.

**Prognosis.** The disease is a serious one, but the large majority of cases recover.

**Treatment Prophylactic.** This is concerned with the destruction of rats and the wearing of shoes by miners in infected areas.

**Curative.** The patient should be put to bed, and the bowels opened with calomel gr 8 and mag sulph gr 120 the following morning. The diet should be liquid, avoiding fats as for catarrhal jaundice (see p 88). Dextrose orangeade should be given and alkalis in doses sufficient to render the urine alkaline. Antispirochaetal serum is available. 20 mls should be given intravenously three times a day for 3 days in a severe case. The faeces and urine must be disinfected as for enteric fever (see p 559).

## Leprosy

**Definition.** A disease characterised by the formation of specific infective granulomata affecting chiefly the skin and subcutaneous tissues, mucous membranes and peripheral nerves.

**Etiology.** Leprosy is caused by the *Mycobacterium lepræ* (B lepræ). It is thought that infection occurs in the majority of cases through the skin less frequently by inhalation. No intermediate agent is known. The disease is thus probably spread by direct contact with an 'open' case of leprosy, which may not have been diagnosed. Nodular cases are more infectious than nervous ones. Children born of lepers develop the disease in over 40% of cases, unless removed from their parents at birth, the disease is not hereditary. **Predisposing causes.** 1 **Locality.** Leprosy is endemic in various parts of the world, especially in India, China, Japan, tropical Africa, the East and West Indies, cases also occur in Europe e.g. in Iceland, Norway, the Balkan States, and in Australia and America. 2 **Unhygienic home conditions and overcrowding.** 3 **Age.** The greatest number of cases show themselves between the ages of 10 and 30 years.

**Pathology.** Granulomata are produced, containing "lepra cells," which may enclose lepra bacilli. The bacilli are found in the skin nodules and ulcers, in the nasal mucous membrane, in the liver and

spleen, in the blood at times during life, and to a lesser extent in the nerves.

**Incubation Period.** In the majority of cases this is between 2 and 5 years.

**Clinical Findings.** The onset is insidious, and before any lesion is apparent there may be prodromal symptoms such as malaise, muscular pains, sweating, with some pyrexia lasting for 7 to 10 days at a time. There are three main types, which will be described separately :

1. *Nodular Leprosy.* When cases are carefully observed the initial lesion is often found to be solitary. It is situated usually on the buttocks or back, or extensor surface of limbs, or on the cheek. The lesion is a small red or reddish-brown, slightly raised shiny spot, about 0.5 cm. in diameter; further crops of spots appear with fever, and on fading they may leave some pigmentation. Some of the spots do not disappear but enlarge to form nodules, and these may ulcerate. Thickening of the skin and face and ear lobes appears, which, together with the nodules, causes a leonine appearance; the outer part of the eyebrows falls out. Nodules may form on the conjunctiva, or in the mouth or larynx, and by ulceration cause blindness, hoarseness, etc. Necrosis of deeper tissues may occur with loss of fingers or toes.

2. *Maculo-anaesthetic Leprosy.* In this variety macules may be seen on the skin in the early stages, 1 or 2 inches in diameter and of varying colour—pink, violet, brown or white. Sensation to light touch is often absent over these areas and they do not sweat. Thickening also occurs in nerves; the ulnar nerve becomes palpable at the elbow and other nerves such as the peroneal or tibial may be felt. Disturbance of their function results in loss of sensation to touch, heat, cold and pain, and muscular wasting, especially of the small muscles of the hand, with a "claw" deformity. The lesions may result in a bilateral facial paralysis. Perforating ulcers occur in the feet.

3. *Mixed Types.* A case may begin as nodular leprosy and later nerve changes occur, or more rarely *vice versa*, or both types may develop simultaneously.

**Differential Diagnosis.** Diagnosis is made in early cases by the appearance of the lesions, and by finding the lepra bacilli. This is effected by cutting out a small portion of the skin and making a smear on a slide from the subcutaneous tissue, or by examining juice obtained from a nodule, or by nasal scrapings. The organism must be differentiated from the tubercle bacillus. The differential diagnosis includes other skin diseases such as erythema multiforme, lupus vulgaris, mycosis fungoides, syphilis, and nerve lesions such as syringomyelia.

**Course and Complications.** The course is usually prolonged for 10 to 20 years or more. The disease passes through phases, such as the early quiescent stage, during which there is a slow multiplication of the bacilli with spread of local lesions; this is followed by a reactionary stage, with rapid proliferation of the bacilli and inflammation of the lesions and constitutional disturbance, and later there is a resolution stage in which the bacilli become granular and break down, and the

local lesions tend to heal. The patient may then present marked deformities, but the disease is arrested. The most important complications are pulmonary tuberculosis and nephritis.

**Prognosis.** The use of preparations of chaulmoogra oil results in the apparent cure of the majority of early cases.

**Treatment.** *Prophylactic.* The incidence of leprosy can be materially lowered by: 1. Isolation of active cases. 2. Examination of home contacts every 6 months for 5 years. 3. Removal of the children of lepers at birth. 4. Treatment of early cases.

*Curative.* Early cases, if not infective, can be treated in clinics, more advanced and infective cases should be isolated. Best results are obtained from injections of a chaulmoogra oil derivative, such as a 3% solution of sodium hydnocepaste (Alepol). When the disease is in the reactionary stage care must be taken with the injections in order to prevent a flare up of the disease, 0.5 ml. is first injected subcutaneously under a lesion and the dose is increased by 0.5 ml. at weekly intervals, up to 4 to 5 mls, provided no general or local reaction occurs. If this does happen the injections should be stopped for a week or so, and a smaller dose given next time. Intravenous injections can next be given of 1 to 2 mls, care being taken to draw some blood back into the syringe, which is mixed with the solution by rotating the syringe with the needle still in the vein, and then the whole is injected. This manoeuvre prevents intravascular thrombosis. The skin lesions may also be treated by X rays and those in the eye or mouth by radium.

## Relapsing Fever

(*Lamine Fever. Tick Fever*)

**Definition.** An intermittent fever due to a treponeme.

**Etiology.** Relapsing fever is caused by a treponeme, of which there are several varieties morphologically similar but which can be differentiated serologically. The average length of the treponeme is  $18\mu$ . The following are the main types. The *Treponema recurrentis* (*Spirillum obermeieri*). This is conveyed to man by the bites of lice (*Pediculus vestimenti* and *capitis*). The treponemes may be excreted in the faeces of the louse or exuded from its body fluid if it is crushed on the skin, and they are inoculated usually by scratching. The treponemes are found in the blood of the patient during the febrile periods of the disease and lice become infective about 10 days after biting such a patient. The type of relapsing fever thus produced is met with chiefly in Eastern Europe and occasionally in North and West Africa, India, Ireland and America. Epidemics occur chiefly in the winter, especially amongst the poor. *Treponema duttoni*. This is conveyed by a tick (*Ornithodoros moubata*). These ticks infest houses and there is no seasonal incidence of the disease. The treponeme causes African tick fever, especially in Eastern and Central Africa and the Congo. Other varieties include *T. carteri* (India), *T. berkhartum* (N. Africa) conveyed by lice, and *T. persicum* (Persia) conveyed by ticks.

**Pathology.** Post mortem the skin is often yellow and petechiae

may be seen. The spleen is enlarged and may contain infarcts. Treponemes are found in the spleen, liver and bone marrow.

**Incubation Period.** 2 to 10 days.

**Clinical Findings.** The onset is usually sudden with shivering or a rigor, severe headache, pains in the eyes, legs and wrists, giddiness, nausea, vomiting and abdominal pain. There may also be epistaxis.

**On Examination:** The temperature rises rapidly to 104° F. or over and the pulse is frequent. There may be slight icterus of the conjunctivæ or definite jaundice, and in some cases a pink macular rash is seen on the neck, trunk and limbs with some petechial hæmorrhages. The spleen is enlarged and there may be tenderness over the liver. The blood: Treponemes are present. There is usually a leucocytosis and the Wassermann reaction may be positive. The adhesion test is also positive, specific immune serum causing the treponemes to adhere to platelets. The temperature remains irregularly raised for about 4 to 6 days and then falls by crisis with sweating. For the next week the patient feels well and is afebrile. A relapse occurs with a sudden rise of temperature and a return of symptoms lasting for about 2 days. Convalescence then generally ensues.

**Varieties.** African tick fever. The onset is usually more gradual, relapses more numerous and the pyrexial period is shorter, 1 to 2 days.

**Differential Diagnosis.** At the onset the disease may be mistaken for such conditions as influenza, malaria, dengue or typhus fever. Diagnosis is established by finding the treponemes in the blood.

**Course and Complications.** The course varies somewhat with the different types. Complications include: Pneumonia, otitis, parotitis, nephritis, hæmatemesis and meningeal symptoms.

**Prognosis.** The disease is a serious one unless adequately treated. The African variety is usually more fatal than that caused by the *T. recurrentis*.

**Treatment.** *Prophylactic.* Lice in the clothes and head should be destroyed and native houses avoided which are infested by ticks.

*Curative.* The patient must be put to bed, kept on a fluid diet, the bowels opened with a saline, and an intravenous injection given of neoarsphenamine, 0.4 G. for a man, 0.3 G. for a woman, and 0.1 G. for a child. It is most effective if injected as the temperature is rising and should not be given immediately before the crisis is expected or severe collapse may result. One injection is usually sufficient to prevent relapses.

### Trypanosomiasis (Sleeping Sickness)

**Definition.** A disease characterised by enlargement of lymphatic glands, followed by nervous changes and increasing torpor.

**Etiology.** Sleeping sickness is due to infection with a trypanosome, a flagellate protozoon. Man is infected by the bite of a tsetse fly. There are two main types: *T. gambiense* conveyed by the fly *glossina palpalis*, occurring in Uganda and the Congo. *T. rhodesiense*, conveyed by the fly *glossina morsitans*, occurring in Rhodesia, Nyassaland, Tanganyika,

and Kenya In tropical America there is a variety caused by the *T. cruzi*. The trypanosome undergoes a cycle of development in the tsetse fly and finally reaches the salivary glands to infect man when he is bitten. The fly may become infected by biting infected big game or man. Rarely the fly will bite a man and carry the infection direct to another man, without any intermediate cycle. *Predisposing causes*  
 1 Locality The disease is endemic in Equatorial Africa and America. *T. gambiense* infections occur near water, and *T. rhodesiense* in dry areas.  
 2 Age Children and adults are infected.  
 3 Race White and black races are equally susceptible.

*Pathology* Post mortem there may be macroscopical evidence of meningitis, or changes only seen microscopically in the meninges over the brain and cord. Meningo encephalitis and meningo myelitis may also occur with perivascular infiltration of lymphocytes. The changes resemble those found in dementia paralytica. The cerebro spinal fluid is under pressure and trypanosomes occur in it. Lymphatic glands are enlarged.

*Incubation Period* This is variable, 2 weeks or longer.

*Clinical Findings* The illness begins insidiously, the patient feeling unwell owing to fever.

*On Examination* During the first stage, the temperature is irregularly raised, the pulse is frequent, the posterior cervical glands are enlarged, there may be some oedema of the legs, and in white people a circinate erythema may be seen on the trunk. The blood. An excess of globulin may be demonstrated in the serum. Trypanosomes may be found in the deposit after centrifugalisation of the blood. The glands. Examination of the juice removed by puncture is more likely to show trypanosomes than is a blood film. The disease, if untreated, gradually passes after months or years into the second stage, characterised by changes in the cerebro spinal fluid. These include an excess of cells (50 or more per c mm.) and excess of protein (over 0.05%). Trypanosomes are not often demonstrable. The patient becomes sluggish mentally and physically, he appears dejected, complains of pains in the body and loss of strength. The glands in various parts of the body become larger. He becomes more sleepy and dozes during the day. Tremors are seen in the tongue and muscles of the arms. The blood now shows an anæmia, and the large mononuclears may be increased to about 10% or over. In the third stage the patient is bed ridden and finally becomes comatose.

*Differential Diagnosis* Malaria is excluded by blood examination and the failure to respond to quinine. Finding the trypanosomes by gland puncture usually establishes the diagnosis in the early stages.

*Course and Complications* The course of an untreated case is as described above, the disease can now usually be arrested by early treatment. Complications include Septic rhinitis, otitis, terminal pneumonia and dysentery.

*Prognosis* This depends largely on early treatment, but infections with the *T. rhodesiense* are more grave than those with the *T. gambiense*.

*Treatment Prophylactic* It has been suggested that big game



should be destroyed in endemic zones, as they harbour the trypanosomes. They may, however, be beneficial, the tsetse flies biting them rather than man. The *glossina palpalis* lives in vegetation near water, and trees should be cleared in these zones. Travellers through such districts should wear gloves and helmets. Spread of the disease can be checked by the compulsory treatment of all natives affected.

**Curative.** Early cases may be treated by the intravenous injection of suramin (B.P. Add.) 1 G. every 3 days for 10 doses. Tryparsamide yields the best results in more advanced cases. It is given intravenously or intramuscularly, 1 to 3 G. in 10 mls of water every week, until a total of 20 to 100 G. has been given, according to the severity of the disease. The average dose is reckoned on a basis of 0.04 G. per kg. of body weight. Visual disturbances are a signal for stopping the injections as optic atrophy may ensue.

### Dengue

#### (Break-bone Fever)

**Definition.** A disease characterised by fever and severe pains in the back and limbs.

**Etiology.** The causative agent is probably an ultra-microscopic and filterable virus which is present in the blood. It is conveyed from man to man by the mosquito *Aedes aegypti* (*Stegomyia fasciata*). The virus undergoes some development in the mosquito, the latter becoming infective in 11 days. The patient's blood is infective for the mosquito during the first 3 days of the illness. Large epidemics and sporadic cases occur. **Predisposing causes :** 1. **Locality :** The disease is met with in New South Wales, Florida, Brazil, the West Indies, Fiji, Syria, Greece, Turkey and Africa. 2. **Climate :** Heat and moisture are required. A previous attack usually confers immunity.

**Pathology.** The disease is rarely fatal, but œdema of the lungs has been noted.

**Incubation Period.** This is usually 5 to 7 days.

**Clinical Findings.** Three stages are described : *The invasion.* The onset is usually sudden with a rigor. The patient complains of aching in the head and eyes and excruciating pains in the lower part of the back and legs which cause complete prostration.

**On Examination :** The conjunctivæ are injected, the face is flushed, the skin generally hot and dry with some erythema. The temperature rises rapidly to 102° F. or higher. The blood shows a leucopenia of about 2,000 white cells per c.mm., owing to diminution of the polymorphonuclears which may fall to 40 or 50%. The urine often contains a trace of albumin.

**The remission.** About the third day sweating occurs, with often nose-bleeding and diarrhœa, the temperature falls by lysis or crisis and the patient feels better. **Terminal fever.** About the fifth day the temperature rises again to 100° F. or so, and remains raised for about 24 hours. There is recurrence of the pains and a rash appears. This is usually morbilliform in character and starts on the palms and backs

of the hands, spreading to arms, trunk and legs It fades in 2 or 3 days, with slight desquamation

**Differential Diagnosis** The occurrence of the disease with its typical course in a warm climate is characteristic It must be diagnosed from other conditions, such as influenza, acute rheumatism, malaria and measles

**Course and Complications** The course is usually as described Complications include Cutaneous boils, periarthritis of the knees or ankles and post-dengue debility

**Prognosis** This is good The disease is practically never fatal

**Treatment Prophylactic** This is concerned with elimination of mosquitoes and prevention of their bites, as for yellow fever (see p 697)

**Curative** The patient should be kept in bed until the rash has gone A saline aperient should be given at the onset Drugs are required for the relief of pain, aspirin in doses of gr 10 six hourly, sodium salicylate gr 15 with sodium bicarbonate gr 30 six hourly, and in some cases a subcutaneous injection of morphin sulph gr  $\frac{1}{4}$  to  $\frac{1}{2}$

## Yaws

(*Frambæsia*)

**Definition.** A specific infective granuloma

**Etiology** Yaws is caused by the *Treponema pertenue* This cannot be distinguished microscopically from the causative organism of syphilis It is not a venereal disease and may be conveyed by direct contagion through a cutaneous abrasion and possibly by insect bites

**Predisposing causes** 1 **Locality** East and West Africa, the Malay States, Philippine Islands, Fiji and Samoa, Burma, Ceylon, Brazil and West Indies 2 **Age** It is not congenital and occurs especially in children and young people 3 **Race** Patients are usually natives

**Pathology.** The "Yaw" is an infective granuloma The disease is distinct from syphilis, but the Wassermann reaction is positive in the majority of cases

**Incubation Period** Probably a month or longer

**Clinical Findings** The onset is insidious with malaise, headache, pains in the muscles and bones, and there may be slight fever The primary lesion may not be detected and may be single or multiple It generally occurs below the knees or near the mouth and is called the "Mother Yaw" It is a papule varying in size up to 2 or 3 inches in diameter, which becomes nodular, exudes fluid and forms a scab The secondary stage Some desquamation of skin occurs in about 3 months' time followed by the appearance of more papules which protrude, become red resembling a raspberry (*frambæsia*) and crust over They may occur in any part of the body and after some weeks eventually drop off They are painless but may itch and treponemes are found in smears Tertiary lesions form from ulceration of the yaw

**Varieties** 1 "Crab" yaws affect the sole of the foot 2 "Gangosa"

is an ulcerating yaw in the palate. 3. "*Goundou*" is a nodular swelling on the nose. 4. "*Juxta-articular nodules*" may form tumours near the knees or elbows.

**Differential Diagnosis.** The disease can be distinguished from syphilis by the absence of a primary genital lesion and the absence of nervous manifestations, such as tabes and general paralysis.

**Course and Complications.** The average duration, if untreated, is about a year, but recurrences of secondary lesions may take place.

**Prognosis.** The disease is not usually fatal.

**Treatment.** Yaws readily responds to injections of neoarsphenamine. For an adult 3 to 6 doses of 0.6 G. intravenously should be given weekly. An intramuscular injection of 0.2 G. of sodium potassium bismuth tartrate in 2 mls of distilled water is also effective. This should be injected weekly for 6 doses.

### Phlebotomus Fever

(*Sand-fly Fever. Papataci Fever. Three-day Fever*)

**Definition.** A specific disease characterised by headache and generalised pains, due to the bite of a sand-fly.

**Etiology.** Phlebotomus fever is believed to be caused by an ultra-microscopic filterable virus which is present in the patient's blood during the first 1 or 2 days of the illness. It is carried by a sand-fly (*Phlebotomus papataci*). The virus probably undergoes changes in the fly's body, as the latter does not become infective for 6 days after biting a patient. *Predisposing causes:* 1. *Locality:* The disease occurs in the eastern part of the Mediterranean, and in Mesopotamia, India and Persia. 2. *Climate:* Warmth and moisture are necessary.

**Pathology.** There are no post-mortem findings.

**Incubation Period.** This is 4 to 7 days.

**Clinical Findings.** The bite of the fly produces some local irritation and swelling. There may be malaise for a day or so before the onset, which is usually sudden with a rigor. The patient complains of frontal headache, pains behind the eyes, in the neck, back and limbs. There is often insomnia.

**On Examination:** The face is flushed and the conjunctivæ are red. The throat is congested and some vesicles may be seen on the mucous membrane. The tongue is furred in the centre. The temperature rises rapidly to 103° or 105° F. but the pulse remains slow, about 70 or 80. The blood: There is usually a leucopenia. The temperature falls in about 2 days and there may be some diarrhoea, epistaxis and sweating. The pulse becomes slower, 40 to 50. There is no rash.

**Differential Diagnosis.** Sand-fly fever must be distinguished from malaria, influenza and dengue. Examination of a blood film excludes malaria; the course of the disease and its occurrence where there are sand-flies usually serve to establish a diagnosis.

**Course and Complications.** The usual course is as described above. Rarely a recrudescence occurs with a rise of temperature to a lesser degree for a short time, about the fifth or sixth day.

**Prognosis** This is good The disease is never fatal

**Treatment Prophylactic** Sand flies should be destroyed and their breeding places eliminated by spraying tar around habitations Long boots should be worn after sunset and electric fans used to keep away the flies An ointment containing oils of eucalyptus anise and turpentine m 3 of each and lanoline oz 1 applied to the wrists and ankles helps to keep away the flies Mosquito nets are not protective as the flies pass through their mesh

**Curative** Liq iodi mitis should be applied to the bites The patient should be kept in bed for 5 or 6 days and the pains relieved by aspirin gr 10 t d s

### Rat-bite Fever

(Sodoku Cat bite Disease)

**Definition** A disease caused usually by the bite of a rat, infected with a special spirillum

**Etiology** Rat bite fever is caused by the *Spirillum minus* (*Spirochaeta morsus muris*) The spirilla live in rats man is infected by their bite or by the bite of an infected ferret, or cat **Predisposing cause** **Locality** The disease occurs in Japan in parts of India such as Bombay, and sporadically in other parts of the world

**Pathology** The lymph glands may be enlarged and hemorrhages seen in the lungs The spleen is enlarged and the spirillum is found in the internal organs

**Incubation Period.** This varies up to about 2 months

**Clinical Findings** The bite wound usually heals but after a week or so breaks down with enlargement of the neighbouring lymphatic glands The patient now complains of headache, aching in the limbs and there may be a rigor with nausea or vomiting

**On Examination** There are periodical rises of temperature up to about 102° F lasting 2 to 5 days followed by a crisis and an afebrile interval of 1 to 2 days A purplish papular rash may be seen on the arms or trunk during the fever Conjunctivitis and small painful swellings in the muscles have been noted The relapses may continue for several months The blood The spirillum may be found in the blood during pyrexial stages There is usually a leucocytosis of about 15 000 per c mm with some eosinophilia and a positive agglutination reaction to the spirillum The blood Wassermann reaction is positive

**Differential Diagnosis** The history of the bite and finding the organism in the blood differentiate rat bite fever from such diseases as relapsing fever and trench fever

**Course and Complications** The course is usually prolonged unless cut short by treatment Nephritis may occur

**Prognosis** The disease is serious unless adequately treated

**Treatment.** Rat bites should be cauterised Intravenous injections of 0.3 G of neoarsphenamine should be given at intervals of five days until the temperature remains normal the leucocytosis disappears and the Wassermann reaction is no longer positive

## Malta Fever

*(Mediterranean Fever. Undulant Fever)*

See also Abortus Fever, p. 559.

**Definition.** A disease due to a specific micro-organism and conveyed by milk.

**Etiology.** The *Brucella melitensis* is the causative organism. Man usually contracts the disease through drinking goat's milk, a large number of the goats in Malta being infected. Water may become contaminated from goat's urine and the disease may also be transmitted through cheese.

The disease is not confined to Malta, but occurs in almost every part of the world. The pathology, clinical findings and treatment closely resemble the account given on p. 560 for abortus fever. In general, Malta fever is a more severe illness.

## Leishmaniasis

A group of diseases due to infection with protozoa of the Leishman-donovan type. The group includes kala-azar, infantile kala-azar and tropical sore. The two former diseases are now considered to be caused by the same protozoon and will be described together.

## Kala-azar

*(Dumdum Fever. Black Fever)*

**Definition.** A disease characterised by enlargement of the spleen, cachexia and irregular fever, due to infection with a specific protozoon.

**Etiology.** The protozoon causing kala-azar is the Leishman-donovan body (*Leishmania donovani*). The protozoon appears in man as a small oval or cockle-shaped body, about  $1.3 \times 3\mu$ , with two nuclei. Although the parasite develops readily in the sand-fly, *Phlebotomus argentipes*, infection of man by the bite of this fly has not been proved. Infantile kala-azar is due to the *Leishmania infantum*, which is probably the same protozoon. **Predisposing causes:** 1. **Locality:** Kala-azar was first described in Assam, it occurs in other parts of India, in China, the Sudan, West Africa, etc. Infantile kala-azar is found around the Mediterranean. It is more prevalent in the country than in towns. 2. **Climate:** Warmth and moisture favour its appearance.

**Pathology.** The Leishman-donovan body occurs in endothelial cells, especially in the spleen; in the blood it may be present in polymorphonuclear leucocytes. A flagellate stage is found in certain sand-flies and can be obtained on culture of the cockle-shaped bodies. Post-mortem, the spleen is enlarged and somewhat firm; the liver is also a little enlarged, and ulcers may be found in the small and large intestine. The marrow of the long bones is unduly red.

**Clinical Findings.** The incubation period is uncertain, varying from a few months to over a year. The disease usually begins insidiously, with progressive weakness, loss of weight, and malaise; there may be diarrhoea, sweating or bleeding from the nose or gums. In some cases the onset is more sudden, suggesting malaria, and in others resembling typhoid fever.

**On Examination** In an established case the patient is wasted, and in Europeans the skin is seen to be pigmented. The spleen is enlarged and the liver may be palpable. The temperature is irregularly raised and may present 2 or 3 summits in the 24 hours. The pulse is proportionately frequent. The blood. There is anæmia and leucopenia is pronounced about 2 000 per c mm. Leishman-donovan bodies are seen at times in polymorphonuclear leucocytes. There is an excess of globulin in the blood. Diagnosis may be confirmed by withdrawing some splenic tissue by spleen puncture, making a film and staining for Leishman donovan bodies. Sternal puncture will also show the Leishman donovan bodies in the monocytes, and this is a safer method of diagnosis than is splenic puncture.

**Differential Diagnosis** Other causes of chronic enlargement of the spleen (see p 518) should be excluded, if there is doubt as to the diagnosis. A blood examination usually excludes malaria, as does the response to quinine treatment. Finding the protozoon by sternal or spleen puncture settles the diagnosis.

**Course and Complications** The average course of the disease, if untreated, is from a few months to 2 years, when death occurs from progressive weakness, or some septic complication, such as pneumonia or gangrene of the lungs.

**Prognosis** The mortality has been lowered by antimony treatment from a previous 96% to about 12%.

**Treatment Prophylactic** Healthy natives in a kala azar district should be kept together at night and away from those infected with the disease. The sand fly range is only a few hundred yards. Nets to afford protection would have to be of too fine a mesh to be tolerated at night.

**Curative** Intravenous injections of trivalent antimony salts such as a 2% sodium antimonytartrate solution in freshly prepared sterile water should be given. The initial dose is gr  $\frac{1}{4}$  for an adult or 1 mg per kg of body weight. If no toxic effect is produced injections are made every fourth day, gradually increasing the dose according to the patient's tolerance until gr  $2\frac{1}{2}$  are reached. The total amount injected in the course should be gr 31. There is often cough at the time of the injection, but if there is nausea or vomiting the next dose should not be increased. The urine should be examined daily for albumin, and if present the treatment stopped. If necessary, another course can be given in 3 or 4 months' time. Pentavalent antimony preparations such as Neostam are very efficient. Ten daily intravenous injections are given, the first dose being 0.1 G, the second 0.2 G and subsequent doses 0.25 G.

### Tropical Sore

(Dermal Leishmaniasis    Delhi Boil    Oriental Sore    Baghdad Boil  
Aleppo Sore)

The causative organism is the *Leishmania tropica*, which cannot be distinguished from the *L. donovani*. Ulceration occurs in the skin and on mucous membranes, the disease is probably spread by a sand fly,

the *Phlebotomus papatasi*. In the American tropics this form of leishmaniasis is called Espundia.

Treatment consist in injections of sodium antimonyltartrate, as for kala-azar (see above), giving about 20 grains in all, or by injections of Neostam.

### Tularæmia

(*Rabbit Fever. Deer-fly Fever*)

**Definition.** A disease characterised by fever and glandular enlargement, due to a specific bacillus.

**Etiology.** Tularæmia is caused by the *Brucella tularensis*. The disease occurs in squirrels, rabbits, water rats and sheep. It is conveyed to man by the deer-fly (*Chrysops discalis*). Man may also be infected in preparing dead rabbits for eating, and in laboratory work. Locality : Tularæmia is met with in Tulare County, California, in other parts of America, Japan, Russia and Norway.

**Pathology.** Suppuration may be found in the lymphatic glands associated with the local lesion.

**Incubation Period.** This is usually between 2 and 5 days.

**Clinical Findings.** *The Glandular Type.* A local papule appears at the site of the fly-bite. This ulcerates and the neighbouring lymphatic glands enlarge. There is irregular temperature, which may last for 2 or 3 weeks. The blood : A positive agglutination test may be obtained towards the end of the second week.

*The Typhoid Type.* When laboratory workers are infected the disease assumes the character of a septicæmia. There is irregular fever lasting for 2 or 3 weeks and relapses may occur for several months. The blood culture is usually positive.

**Differential Diagnosis.** The disease has to be distinguished from other septicæmias and causes of glandular enlargement with fever, such as the enterica group and plague. Injection into a guinea-pig of the fluid obtained by gland puncture is a method of isolating the bacillus.

**Course and Complications.** The course is as described above. Bronchitis, bronchopneumonia, pneumonia or pleural effusion (containing the *Br. tularensis*) may occur as complications.

**Prognosis.** The disease is not fatal.

**Treatment.** The patient must be kept in bed and the glands opened when they suppurate. Good results have been obtained in some cases of tularæmic pneumonia by the use of antitularæmic serum.

## CHAPTER XIII

### THE PARASITIC WORMS

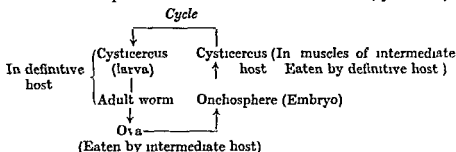
#### THE CESTODES

##### (Tape Worms)

**Definition** Segmented flat worms, usually hermaphrodites

**Anatomy** The tape worms consist of a head and neck (scolex) and segments (proglottides) The segments near the head are immature, the intermediate ones contain sexual glands and the terminal ones ova They have no alimentary canal

**Varieties** The most important varieties are the *Tænia solium* the *Tænia saginata*, the *Diphyllbothrium latum* (*Dibothriocephalus latus*), the *Echinococcus granulosus* (*Tænia echinococcus*) and the *Hymenolepis nana* (*Tænia nana*) Man is infected by eating the larvæ in meat or fish which is raw or insufficiently cooked The larva is known as a cysticercus if it forms one cyst and one scolex (as in *T. solium* and *T. saginata*), and as an echinococcus if it forms many cysts and many scolices in each cyst (as in *E. granulosus*) The larva of the *D. latum* is a plerocercoid and elongated like a worm The larva develops in the definitive host into the adult worm, and the eggs which pass out in the feces are eaten by the intermediate host The outer layer is dissolved in the alimentary canal and the contained embryo (onchosphere) is liberated This passes into the tissues to form the larva (cysticercus)



The ovum of the *D. latum* forms a ciliated embryo, this is eaten by certain crustacea (such as *cyclops strenuus*) and forms a proceroid larva The cyclops containing the proceroid is eaten by a fish and becomes encysted as a plerocercoid larva Man eats the fish, and is infected the larva developing into the adult worm In *E. granulosus* infection man eats the ova and the larva formed develops in his tissues, the adult form occurring in animals such as the dog

#### The *Tænia Solium*

(The Solitary Tape Worm The Armed Tape Worm The Pork Tape Worm)

The chief features are as follows

**Infection** By eating imperfectly cooked "measly" pork containing the larvæ (*Cysticerci cellulosa*)



**Locality.** World-wide distribution.

**Hosts.** Definitive: Man. Intermediate: The pig or rarely man (*Somatic taeniasis*).

**Length of Adult Worm.** About 10 feet.

**Head.** 1 mm. in diameter. Globular, with 4 suckers and a rostellum armed with 2 rows of 14 hooklets. Neck, thin.

**Segments.** About 1,000. Contain a uterus with about 10 lateral branches; the genital pore is lateral and alternates regularly. Mature segments are three times as long as they are broad (see Fig. 67).

**The Ova.** Circular. Diameter about 35  $\mu$ .

**The Onchosphere (embryo).** About 20  $\mu$  in diameter. Has 6 hooks.

**The Cysticercus Cellulosæ.** Like

a little bladder; oval, about 5 to 20 mm. long. Single scolex forms in it.

Segments containing ova are passed in man's feces and are eaten by the pig. Rarely *somatic taeniasis* occurs in man, either by transference of ova by his fingers to the mouth, or owing to segments containing ova being regurgitated into his stomach. The cysticerci (bladder worms) may then develop under the skin as small nodules, or in the brain, causing epilepsy, in the eyes, or muscles (see Fig. 68). They may be revealed by X-ray examination, and must not be confused with the cysts of the *Trichinella spiralis*. The latter are probably too small to be seen radiologically.

### The *Tænia Saginata*

(*Tænia Mediocanellata*. The Unarmed Tape Worm. The Beef Tape Worm. The Fat Tape Worm)

**Infection.** By eating imperfectly cooked beef infected with the *Cysticercus bovis*.

**Locality.** World-wide distribution.

**Hosts.** Definitive: Man. Intermediate: The ox.

**Length of Adult Worm.** About 20 feet.

**Head.** 2 mm. in diameter, Pear shaped, with 4 suckers, but no rostellum and no hooklets. Neck, medium size.

**Segments.** About 2,000. Contain a uterus with about 20 lateral

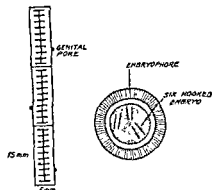


FIG. 67. *T. SOLIUM*. MATURE SEGMENTS. (Natural size.)

*T. solium*: fecal ovum (onchosphere), brown, circular, 35  $\mu$  in diameter. ( $\times 500$ .)

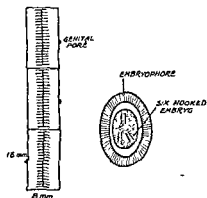


FIG. 60. *T. SAGINATA*. MATURE SEGMENTS. (Natural size.)

*T. saginata*: fecal ovum (onchosphere), brown, oval, 38  $\mu \times 23 \mu$ . ( $\times 500$ .)

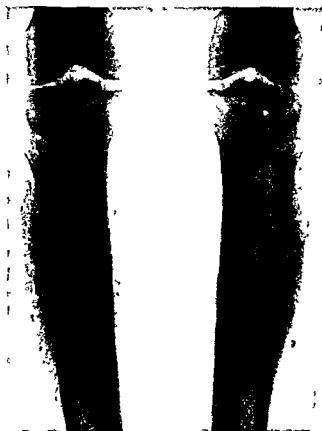


FIG 68 SOMATIC TENIASIS CYSTICERCI IN MUSCLES  
OF LEGS.

ranches, the genital pore is lateral and alternates irregularly, the mature segments are twice as long as they are broad (see Fig 69)

The Ova Oval, measuring  $38\ \mu$  by  $25\ \mu$  approximately

The Onchosphere (embryo) Head has 6 hooks

The Cysticercus Bovis 8 mm by 5 mm Single scolex forms in it

The segments containing ova are passed in man's feces and are eaten by the ox

### The Diphyllobothrium Latum

(The Dibothriocephalus Latum The Russian Tape Worm)

Infection. By eating caviare or imperfectly cooked fish

Locality Russia, the Baltic Switzerland

Hosts Definitive Man Intermediate Fish such as pike perch, salmon trout and grayling

Length of Adult Worm About 30 feet

Head. 3 mm long Olive shaped with a suction groove on its dorsal and ventral sides There are neither rostellum nor hooklets

Segments About 3,000 Contain a central rosette uterus and a ventral genital pore They are broader than they are long (see Fig 70)

The Ova Oval measuring about  $60\ \mu$  by  $40\ \mu$  with an operculum

The Onchosphere Ciliated and free swimming It is eaten by crustacea (such as the *Cyclops strenuus*)

The Proceroid Larva Elongated oval  $5\ \mu$  long

The Plerocercoid Larva Worm like about 6 mm long Man passes free ova in his feces

Clinical Findings Infestation of man with *T. solium*, *T. saginata* or with *D. latum* may give rise to vague symptoms of indigestion or to marked hunger with bodily wasting A severe anemia of the pernicious type rarely occurs with *D. latum* infestation It is estimated that in Finland 250 000 people are infested but only 0.5% suffer from anemia

Treatment Prophylactic Meat and fish must be adequately inspected for cysticerci and properly cooked

Curative The patient is kept in bed and starved for 2 days During this time only fluids are allowed, such as milk and meat extracts An initial dose of castor oil fl oz  $\frac{1}{2}$  is given and this is followed by mag sulph gr 60 t.i.d. on the second day On the third day no food is allowed until the worm is passed and the patient is kept recumbent Mag sulph gr 120 is administered at 7 a.m., and after the bowels have acted ext siliis liq m 15 in a capsule is given at 9 a.m., at 9.15 a.m., at 9.30 a.m., and at 9.45 a.m., and m 30 at 10 a.m. followed at noon by fl oz 2 of mist sennae co (B.P.) All the actions of the bowels must be received into a bed pan containing a little warm water, and if the head of the worm is not seen the evacuations should be strained through black muslin If the head does not come



FIG 70 THE DIPHYLLOBOOTHRIUM LATUM MATURE SEGMENTS (Natural size.)

Diphyllobothrium latum fecal ovum brown oval  $60\ \mu$   $\times 40\ \mu$  ( $\times 250$ )

away, fresh segments will appear in the motions in about 8 months' time. A second treatment should then be given.

### The Echinococcus Granulosus

(*The Tania Echinococcus*)

**Infection.** Man is infected by ova excreted by dogs, if the dog licks the man's hands or his dishes. Infection may also occur from drinking water or from eating uncooked vegetables contaminated by the ova. The dog is infected by eating meat containing the hydatid cysts.

**Locality.** Australia and Iceland.

**Hosts.** Definitive: The dog, wolf or jackal. Intermediate: Man, sheep, oxen and pigs.

**Length of Adult Worm.** About  $\frac{1}{4}$  inch.

**Head.** Resembles that of *T. solium* in miniature, being 0.3 mm. in diameter. There are 4 suckers and a rostellum with 2 rows of about 20 hooklets in each.

**Segments.** Four. The terminal segment is mature and contains ova.

**The Ova.** Oval, measuring about  $35\mu$  by  $25\mu$ . The ovum, when swallowed by man, liberates its six-hooked embryo (onchosphere); this migrates to the tissues, especially the liver, and forms a hydatid cyst.

**The Hydatid Cyst.** There is an inner granular or germinal layer, from which the daughter and grand-daughter cysts form. These contain many scolices; the middle layer is laminated; the outer layer is fibrous and formed from the tissues of the host. The contents are clear fluid, specific gravity about 1,000. The fluid contains 1% of sodium chloride, a trace of sugar, some hooklets and no protein. The cyst occurs especially in the liver, but may be found in the lungs, brain, kidneys or heart.

### Hydatid Disease

**Clinical Findings.** The patient does not usually notice any ill health unless the hydatid cyst causes mechanical pressure symptoms or some complication occurs.

**Hydatid of the Liver.** If the cyst is deep-seated no tumour is felt, but there may be a palpable swelling which sometimes fluctuates. More rarely the hydatid thrill is felt by placing one hand over the swelling and tapping it sharply with the fingers of the other hand (see p. 77).

**Hydatid of the Lungs.** This is described on p. 182.

**Hydatid of the Kidneys.** This may cause a renal tumour (see p. 481).

**Hydatid of the Brain.** This causes symptoms of a cerebral tumour.

**Hydatid of the Heart.** This may cause sudden death.

The blood may show an eosinophilia and the Casoni intradermic test is positive (see p. 77).

**Course and Complications.** The cyst may grow to 5 or 6 inches in diameter; it may atrophy, calcify or its contents may suppurate. Rupture may occur into the peritoneum, the stomach, intestines, the

pleura, the inferior vena cava or the bile ducts. Rupture may be accompanied by shock and by urticaria, and in cases of intraperitoneal rupture secondary cysts may form.

**Prognosis.** The disease is serious. Frequently, however, the cyst dies and inspissates. Suppuration or rupture make the outlook more grave.

**Treatment.** There is no medical treatment. If possible, the cyst should be removed surgically without rupture.

### The *Hymenolepis Nana*

(*The Taenia Nana* The Dwarf Tape Worm)

This worm infests especially children in America, Egypt and Southern Europe. It is  $\frac{1}{2}$  inch long (12.5 mm) and  $\frac{1}{16}$  inch (1 mm) broad. It has a rostellum with 4 suckers and a single row of about 30 hooklets. There are about 200 segments. There is probably no intermediary host, the eggs which are liberated in the intestine developing into adult worms.

**Treatment.** Oil chenopodii should be given. The dose for children is one drop for each year of age on a lump of sugar, followed 2 hours later by a dose of castor oil appropriate to the age of the child.

## THE TREMATODES

(Flukes)

The trematodes are flat non segmented leaf like worms, usually hermaphrodites, possessing a mouth and 1 or more suckers. The most important varieties which are parasitic in man are the schistosoma and the paragonimus (lung fluke), the fasciola or liver fluke very rarely infests man.

### Schistosomiasis

(Bilharziasis)

There are three varieties

#### Urinary Schistosomiasis

(Endemic Hæmaturia)

**Etiology.** Endemic hæmaturia is caused by the *Schistosoma hæmatobium* (*Bilharzia hæmatobium*). Man is infected by cercariæ (young swimming forms of the trematode) which pierce his skin when bathing or paddling, and infection may occur by drinking water. The cercariæ pass to the portal vein and in about 6 weeks develop into the adult male and female forms of the *S. hæmatobium*. The male is flat, about 15 mm by 1 mm, and the female is slender and round, about 25 mm (1 inch) by 0.25 mm. They migrate against the blood stream from the portal to the pelvic veins and veins of the bladder or rectum, the male curves from side to side to form a gynæcophoric canal in which he embraces the female; when they have reached the small veins, the female leaves the male and migrates to the smallest vein allowing her passage. The ova are then extruded. The ovum is about 100  $\mu$  by 70  $\mu$ , and has a sharp terminal spine; this penetrates the blood vessel,

and the ovum is pushed through the vessel by the blood stream. It then ulcerates its way into the submucous tissues and through the mucous membrane of the bladder or rectum. The ova are chiefly passed in the urine. On coming in contact with water, an embryo or ciliated miracidium hatches out. This enters a spiral fresh-water snail, such as the *Bullinus contortus*. Sporocysts form and cercariæ are liberated into the water from the snail, completing the cycle. The disease is met with in Africa, Arabia and Iraq.

**Pathology.** Post-mortem, the mucous membrane of the bladder is red, thickened in patches and polypi may be seen. The muscular wall is hypertrophied. The prostate may be enlarged and the ureters dilated. Ova may be seen in the submucous tissues of the bladder or in the prostate; the liver may be cirrhotic.

**Incubation Period.** 1 to 3 months.

**Clinical Findings.** The first symptoms appear a few weeks after infection. They constitute the toxæmic stage. The patient complains of urticaria, malaise, with perhaps abdominal pain.

**On Examination:** The temperature is raised and the blood shows an eosinophilia which may reach 50%. A positive complement fixation may be obtained by using as an antigen an extract of the liver of infected snails.

Local symptoms may not be noticed for a few months, or even years. The patient then complains of passing blood, usually at the end of micturition, with perhaps frequency, scalding, and pain in the perineum or suprapubic region. He may also pass blood and mucus in his motions.

**On Examination:** The patient is often pale or sallow. The urine: The deposit shows ova, red corpuscles and pus cells. The blood may show a leucocytosis of about 15,000 per c.mm., with an eosinophilia of about 12%. The complement fixation test is usually still positive. Cystoscopy may reveal an inflamed vesical mucous membrane, and white patches may be seen due to submucous ova.

**Differential Diagnosis.** Other causes of hæmaturia, such as a calculus, cystitis, etc., must be excluded. The presence of ova in the urine is diagnostic, but a calculus may also be present.

**Course and Complications.** The disease, if untreated, pursues a chronic course for many years, and, if the patient is not destroyed by complications, spontaneous cure usually results. Complications include: Cystitis, vesical calculus, hydronephrosis, pyonephrosis, carcinoma of the bladder, urethral fistula or stricture, etc.

**Prognosis.** This depends upon the severity of infection, the presence of complications and the institution of adequate treatment.

**Treatment.** *Prophylactic.* Bathing or paddling in endemic areas should be forbidden, and drinking water should be boiled. The snails may be destroyed by cutting off the water of the irrigation canals periodically for 2 weeks during the dry season. Carriers should have their infestation cured by treatment.

*Curative.* Tartar emetic (antimon. et pot. tartras) is injected slowly into a vein. The injection should be given about 2 hours after

a meal, beginning with gr  $\frac{1}{2}$  dissolved in 2 mls of freshly distilled water, and increasing by gr  $\frac{1}{2}$  at a time until gr  $2\frac{1}{2}$  (10 mls) are given. The injections are made 3 times a week, and a total amount of gr 30 is usually required. The hematuria may be temporarily increased. The urinary deposit should be examined daily for ova, which, as they are killed, shrivel, darken, and when placed in water will not hatch out. A cough is often noticed during the injection. This is due to the antimony reaching the lungs. If toxic symptoms follow an injection, the next dose should not be increased. Toxic symptoms include tightness of the throat or chest, abdominal pain, and a metallic taste in the mouth. If there is collapse at the injection, Pituitrin (ext pit liq B.P.) 1 mil or liq adrenal hydrochlor 0.5 mil should be injected intramuscularly. If the patient has heart or kidney disease, the antimony is contraindicated. When the veins are difficult, stibophen, also a trivalent antimony preparation, may be injected intramuscularly, using a 6.3% solution. Nine injections are given on alternate days. For an adult the dose is 1.5 mil, followed by 3 mls, the subsequent doses being 5 mls.

### Intestinal Schistosomiasis

**Etiology** This disease is caused by the *Schistosoma mansoni* (*Bilharzia mansoni*). The adult trematodes are found in the mesenteric veins, and their ova pass through into the rectum. The ova have a lateral sharp spine. The intermediate host is a flat fresh water snail, the *Planorbis boissyi*. The life history resembles that of the *S. haematobium*. Intestinal schistosomiasis occurs in Egypt, the Belgian Congo, Nigeria, the West Indies and the northern part of South America.

**Pathology** Post mortem, the mucous membrane of the rectum and colon is thickened, and there are papillomatous swellings. The liver may be fibrotic ("pipe stem" cirrhosis) and ova are found in it.

**Clinical Findings** An early toxæmic stage with fever and urticaria occurs, followed in about 2 months' time by dysenteric symptoms, with blood and mucus in the motions. The ova are found in the feces and the blood shows an eosinophilia and gives a positive complement fixation test. In Egypt a special type with enlargement of the liver and spleen occurs. Anemia and ascites are present. This variety is called Egyptian splenomegaly.

**Treatment** This is by tartar emetic or stibophen as for urinary schistosomiasis (see above).

### Eastern Schistosomiasis

**Etiology** Eastern schistosomiasis is caused by the *Schistosoma japonicum* (*Bilharzia japonica*). The trematodes are found in the mesenteric veins, and the ova pass into the large intestine. The ova have no spines but a small lateral knob may be seen. The intermediate host is the *Hemibia japonica*, a fresh water mollusc. The life history resembles that of the types described above. The disease is met with in China, South Japan, the Philippine Islands and Upper Burma.

**Pathology** Post mortem, the liver is enlarged, cirrhotic and

contains ova ; the spleen is enlarged, but usually free from ova ; ova may be found in the brain. The mucous membrane of the large intestine is thickened and polypi may be seen.

**Clinical Findings.** An early toxæmic stage occurs with fever, urticaria and eosinophilia. Later, there are abdominal symptoms, with pain and dysentery.

**On Examination :** The liver and spleen are enlarged and there may be ascites. The ova are found in the fæces.

**Treatment.** This consists in giving injections of tartar emetic or stibophen, as described above (see p. 716).

### Paragonomiasis

**Etiology.** Paragonomiasis is caused by the *Paragonimus westermani* (*Distoma ringeri*). The fluke is about  $\frac{1}{2}$  inch (8 mm.) long.

**Pathology.** The parasites occur in the lungs.

**Clinical Findings.** The flukes are the cause of endemic hæmoptysis (see p. 176).

### Fascioliasis

**Etiology.** Fascioliasis is caused by the *Fasciola hepatica* (*Distoma hepaticum*).

**Pathology.** The flukes are found chiefly in the livers of sheep. Very rarely man is infected.

## THE NEMATODES

These are cylindrical non-segmented, unisexual worms. The following are the most important varieties occurring in man : *Ascaris lumbricoides*, *Enterobius* (oxyuris) *vermicularis*, *Trichuris trichiura* (*trichocephalus dispar*), *Trichinella spiralis*, *Strongyloides stercoralis*, *Ankylostoma duodenale*, *Necator americanus*, *Wucheria* (*filaria*) *bancrofti*, *Loa loa*, *Dracunculus medinensis* and the *Onchocerca volvulus*.

### Ascariasis

#### (Round Worm Disease)

**Etiology.** Ascariasis is caused by the *Ascaris lumbricoides*.

**The Adult Worm.** Male, average size 20 cm. (8 inches) in length and 0.3 cm. ( $\frac{1}{8}$  inch) in diameter. Female, average size 30 cm. (12 inches) in length and 0.5 cm. ( $\frac{1}{2}$  inch) in diameter. They are light brown in colour with pointed ends, and live in the small intestine in man and in animals such as the pig. Their eggs, which measure about 70  $\mu$  by 60  $\mu$ , are passed in the fæces. Man is infected by swallowing the eggs in water or contaminated uncooked food-stuffs. When ingested, the larvæ, which have developed inside the eggs, pass out and are believed to make their way to the liver and lungs, then to the trachea, larynx, œsophagus stomach and intestine, where they develop into adult worms. Infestation is more common in children than in adults.

**Pathology.** Post-mortem, the worms are found chiefly in the upper



the liver, lungs, trachea and œsophagus to the intestines. The worms are said to pierce the mucous membrane with their fine extremity which prevents their being dispelled by anthelmintics.

**Clinical Findings** The worms usually produce no symptoms, but may cause appendicitis. The ova are found in the fæces.

**Treatment** *Ol chenopodii* may be given, for dosage see pp 715, 724.

### Trichiniasis

#### (*Trichinosis Trichinelliasis*)

**Etiology** Trichiniasis is caused by the *Trichinella spiralis*.

**The Adult Worm** The male, average length 1.6 mm, diameter 0.04 mm. The female, average length 4 mm, diameter 0.06 mm. Man is usually infected by eating raw or partially cooked ham, pork or pork sausages, containing encysted trichinellæ. Pigs are infected either by eating raw garbage containing pieces of infected pork or by eating infected rats. The former method is very prevalent in America. Larvæ develop from the cysts and form adult worms which live in the small intestine. The larvæ produce embryos which pass by lymphatics or blood vessels to striated muscles and encyst coiled up near the tendinous insertions of the muscles. They are found especially in the intercostal muscles, diaphragm, the muscles of the neck, upper arm and calf. Calcification may occur in the cyst wall. The disease is common in America and in Germany.

**Incubation Period** This varies from 5 to 10 days.

**Clinical Findings** Three stages were originally described. *The Invasion Stage* This is characterised by malaise, vomiting and watery diarrhoea, with fever and at times delirium. *The Migration Stage* During the second week of the illness pains occur in the arms and legs. There may be difficulty in breathing or in mastication. An urticarial rash may also appear, with œdema of the legs and face. The blood shows a leucocytosis of about 20,000 per c mm, with an eosinophilia of about 10%. Adult worms are rarely found in the fæces. *The Incystment Stage* This usually gives rise to no symptoms.

Epidemics occurred in England in 1941 in which the preliminary gastro-intestinal symptoms were very slight and in some cases constipation was a prominent feature. The onset was characterised by fever of 101° to 102° F, swelling of the eyelids, face and occasionally limbs and frontal headache. "Splinter hæmorrhages" were seen under the nails. In some cases meningeal or encephalitic symptoms were present and in about half the cases a dry cough was an initial symptom. Muscular pains were noted about 3 days after the onset, and temporary mental changes, such as melancholia were not infrequent. The urine was usually free from albumin and the blood showed an eosinophilia of about 40%. Asymptomatic infestation also occurs in which no symptoms of the disease are manifested during life, but post mortem encysted worms are found in the diaphragm.

**Differential Diagnosis** Trichiniasis may be mistaken for acute nephritis or sinusitis owing to the orbital œdema. The muscular pains may suggest acute rheumatism. The nervous symptoms may lead to a

diagnosis of meningitis or of encephalitis. The fever and gastrointestinal symptoms may resemble those of food poisoning or enterica group infections. The initial fever and cough may cause confusion with influenza or bronchitis. The correct diagnosis is suggested by the fever, leucocytosis and eosinophilia. It is established by finding the larvæ in the blood during the first 2 or 3 weeks of the illness. For this purpose 5 c.c. of blood are laked with 10 c.c. of water, and the centrifuged deposit is examined microscopically. The adult worms are seldom found in the fæces. Muscle biopsy will demonstrate the presence of *T. spiralis* in the majority of cases after the fourth week of the illness; a small portion of the pectoralis major, deltoid or gastrocnemius muscle is examined. Intradermic injection of a saline suspension of powdered larvæ gives a positive reaction, as shown by the formation within 5 minutes of a wheal and an erythematous zone, in nearly all people for 6 months after infestation. Reports that calcified cysts can be demonstrated radiologically in the muscles in long-standing cases are probably incorrect. The cysts are usually too small to be thus revealed, and confusion has arisen by mistaking encysted cysticerci for them (see p. 712).

**Course and Complications.** The feverish stage may last for 2 to 3 weeks, and the muscular pains and weakness for several months. Death may occur from myocarditis or encephalitis.

**Prognosis.** In an outbreak in Berlin in 1930, 10 out of 70 cases proved fatal. The mortality was 16% in a series of 1,550 cases in America, and no deaths occurred in 500 cases in the Wolverhampton neighbourhood in the English epidemic of 1941.

**Treatment. Prophylactic.** Infected meat must be avoided, and ham, pork, and pork sausages should be well cooked.

**Curative.** During the invasion period the patient should be given calomel gr. 3, followed by mag. sulph. gr. 120 in the morning or castor oil fl. oz.  $\frac{1}{2}$ , to drive out the worms before the embryos are formed. This treatment should be repeated again the next day. There are no measures available for eliminating the cysts from the muscles.

### Strongyliasis

The *Strongyloides stercoralis* lives in the submucous tissues of the small intestine in man. The female worm measures 2.5 mm. by 0.3 mm. The male is not found in the intestine. It is thought that the worms cause diarrhoea at times (Cochin-China diarrhoea).

### Ankylostomiasis

(*Uncinariasis. Hookworm Disease. Miner's Anæmia*)

**Etiology.** Ankylostomiasis is caused by a small nematode, of which there are two types, the *Ankylostoma duodenale* and the *Necator americanus*.

The *A. duodenale*. The male is about 9 mm. by 0.5 mm. in size and the female slightly larger. Its name implies that it has a bent mouth which contains 4 teeth. It lives chiefly in the jejunum, attached

by its mouth to the mucous membrane from which it sucks blood. Many eosinophil cells congregate at the site of its attachment.

**The *N. americanus*** This is slightly smaller. Thousands of eggs are formed, which are excreted from the human intestine. When these come in contact with damp soil or water, embryos hatch out and enter the skin of man, usually through the feet, producing "ground itch". They then pass in the circulation to the heart and lungs. They burrow into the bronchi and are carried with the mucus up the trachea and pass down the oesophagus into the small intestine. **Locality** The disease occurs throughout the sub-tropical world, that due to the *Ankylostoma duodenale* being met with in Egypt, India, Ceylon, South China, Queensland and the Southern States of North America. It has also been found amongst tin miners in Cornwall, coal miners in Belgium and the workers in the St. Gothard tunnel. It is a disease of the country rather than of towns, and affects children and adults. The *N. americanus* is found in America and also in Central Africa, India, the Philippines, Ceylon, etc.

**Pathology** Post mortem, the body is well nourished, but pale. The heart, liver and kidneys are fatty, and localised hæmorrhages are seen in the mucous membrane of the small intestine. Serous effusions may be present. Over a thousand worms may be found in the intestine.

**Clinical Findings** The local lesions in the feet ("ground itch") result in the formation of vesicles and pustules, which heal in a week or so. It is several months before the general symptoms are felt. The patient complains of progressive weakness, shortness of breath and palpitations. There may also be flatulence, and constipation or diarrhoea. The appetite is good, but pica (dirt eating) is a feature of the disease.

**On Examination** The complexion is pale or sallow. The temperature may be slightly raised. There may be œdema of the feet with dilatation of the heart. The spleen is not enlarged. The blood. The anæmia is of a microcytic hypochromic type. The characteristic feature is an eosinophilia of about 20%. The stools usually show blood either by naked eye or by occult blood tests. Ova are seen microscopically.

**Differential Diagnosis** Other causes of anæmia are excluded by the eosinophilia and the presence of the ova in the fæces.

**Course and Complications** The disease pursues a prolonged course if untreated and results in much economic loss amongst workers in the tropics. Growth is much diminished in infected children. Dysentery with blood and mucus in the motions, polyarthritis or nephritis may occur.

**Prognosis** This is usually good but at times the disease is rapidly fatal.

**Treatment** *Prophylactic* Latrines should be provided and their usage enforced in endemic zones and the feet protected by boots. Drinking water should be boiled.

*Curative* Various anthelmintic drugs are employed. Thymol is given as follows. The patient is kept on a liquid diet for a day or so

and given a saline purge such as mag. sulph. gr. 240 in the evening. The next morning, after the bowels have opened and before any food is taken, finely ground thymol gr. 15 mixed with an equal quantity of lactose is given in a cachet every hour for 4 doses. If the bowels are not opened in 2 hours the saline purge is repeated. No alcohol or chloroform must be taken during the treatment, or the thymol will be absorbed and produce toxic effects. If ova are still found in the stools the treatment is given again in a week's time. Caprokol is safer for small children and for debilitated adults. The method of administration is described on p. 719.

Oil of chenopodium is also used. No purge is given before the treatment. In the morning ol. chenopodii m. 8 is given in a gelatin capsule every hour for 3 doses. Two hours later mag. sulph. gr. 240 is given.

Carbon tetrachloride is dangerous, as if impure it may produce toxic symptoms resembling delayed chloroform poisoning, due to degeneration of the liver. No preliminary starvation or purge is required. The dose for an adult is m. 60, usually given in milk, or in two 30 minim capsules. For children the dosage is m. 3 for each year of age. Mag. sulph. gr. 240 should be given 2 hours later. The safest method of using carbon tetrachloride is to give Tetraform m. 50 in ol. ric. or liq. paraffin fl. oz.  $\frac{1}{4}$ . The mixture must be well shaken before being taken. No purgative is required subsequently. If toxic results ensue dextrose should be given by mouth or intravenously with alkalis, as for acute yellow atrophy of the liver (see p. 89).

### Filariasis

**Etiology.** There are several types of these small nematodes which produce disease in man, the most important being the *Wucheria* (filaria) bancrofti, the *Loa loa* and the *Onchocerca volvulus*.

#### *Wucheria bancrofti*

##### (*Filaria bancrofti*)

Infection is conveyed to man by mosquito bites, chiefly by the *Culex fatigans*. The female mosquito introduces the embryos into human beings, and they pass to the lymphatic vessels and glands, where they develop into the adult filaria. The male is about 40 mm. (1½ inches) long and the female twice its length. They are very fine and resemble a coiled hair. The female produces large numbers of embryos (microfilariae), which pass into the blood stream. They measure about 0.3 mm. long by 7.5  $\mu$  wide. These are taken up by a biting mosquito, and, after undergoing changes in its body, are injected again into man. The embryos are practically absent from the peripheral circulation of man during the day, being located then chiefly in the lungs and kidneys. They pass into the peripheral circulation during the night, beginning to migrate before the patient goes to sleep, reaching their maximum about midnight and diminishing in numbers before he awakes. This corresponds with the night activity of the mosquito. It has been suggested that the migration is caused by a sleep-producing

**hormone** If the patient sleeps by day, the periodicity is altered. In the Pacific Islands the *W. bancrofti* shows no periodicity, the intermediary mosquito, the *Aedes variegatus*, there biting during the day. The disease occurs in the tropical portions of North and South America, the West Indies, in North Africa, Central Africa, China, Japan, North Australia, India and the Malay Archipelago.

**Pathology** The changes produced by the adult filariæ result from lymphatic obstruction and secondary inflammation of lymphatics. In tropical elephantiasis adult filariæ can often be found in the neighbouring lymphatic glands. The lymphatic glands and vessels become fibrosed and the embryos cannot pass through them into the circulation. The embryos are not known to produce any pathogenic effects.

**Incubation Period** This is probably a matter of 4 or 5 years.

**Clinical Findings** The results produced by the adult worms are variable, and include—**Lymphangitis**. Painful red lines form under the skin and the neighbouring lymphatic glands are enlarged. There is constitutional disturbance and fever, and probably a secondary streptococcal infection of the lymphatics. **Elephantoid fever**. Periodical attacks of fever with an initial rigor and terminal sweating occur, somewhat resembling malaria. The deep lymphatic glands are probably inflamed. Other local results of lymphangitis include inflammation of the spermatic cord, testicle and synovial membranes. **Lymphatic varices**. These may affect the glands, especially in the groin (varicose groin glands). They diminish in size on pressure. **Lymph scrotum** is characterised by enlargement of the scrotum with lymphatic varices. **Ruptured lymphatic varices**. Chyluria results from rupture in the urinary tract. The urine is milky and may contain blood. Chylous effusions may form in the peritoneum or in the tunica vaginalis. The blood. There is an eosinophilia. The embryos can usually be seen in a wet film taken at night. **Elephantiasis**. In this condition there is a marked solid œdema, due to lymphatic obstruction. Microfilariae are absent from the blood. The legs are usually affected, there being an enormous swelling with thickening of the skin and subcutaneous tissues. The scrotum may be involved, forming a tumour weighing over 50 lbs. The vulva and arms are less commonly affected.

**Course and Complications** The disease pursues a chronic course. Cutaneous carcinoma may occur as a complication of elephantiasis of the leg.

**Treatment** *Prophylactic*. Mosquito breeding places should be sterilised, and electric fans and mosquito nets used as for malaria (see p. 681).

*Curative*. No specific drug is known, but some favourable results have been obtained with intravenous injections of 2 mls of a 2% solution of antimon et sod tartras (approximately gr  $\frac{1}{2}$ ) increasing the injections gradually to gr 2, at intervals of 4 to 7 days, the total dosage being gr 30 to 35, as for kala azar (see p. 709). The effects produced, however, are not very lasting.

**Acute lymphangitis**. The limb should be rested and elevated, and

a lotion applied, such as Liq. plumb. subacetat. dil., 4 parts, alcohol 90%, 4 parts, aquam ad 100 parts. Sulphanilamide should be given for secondary streptococcal infections, in doses of 1 G. 3 or 4 times a day for 5 days.

Chyluria: Fat should be omitted from the diet.

Elephantiasis: An elastic stocking should be worn, and some promising results have been obtained with protein shock therapy, injecting intravenously 50 millions of T.A.B. vaccine at weekly intervals. Operation may be required for elephantiasis of the scrotum.

### Loa Loa

The adult worm called *Loa loa* is 30 to 35 mm. (1½ inches) long and is slightly thicker than *W. bancrofti*. The embryo is called the *Microfilaria loa* (diurna), and is about the same size as that of the *W. bancrofti*. It appears in the peripheral blood during the daytime, and this periodicity cannot be changed by altering the sleeping hours of the patient. Man is infected through the bites of mangrove flies, such as the *Chrysops*. The adult worm produces in man oedematous swellings under the skin (Calabar swelling of West Africa), which take about 3 days to come and go, recurring irregularly. The worms also pass under the skin, and may invade the conjunctiva or anterior chamber of the eye.

Treatment. No specific treatment is known.

### Onchocerca Volvulus

This microfilaria is smaller than the *W. bancrofti*. Man is infected by the bite of the buffalo gnat, *Simulium damnosum*. It is met with in West and East Africa and in South America. The chief lesions are subcutaneous nodules, iritis and blindness. The treatment is as for *W. bancrofti*; by fever therapy and injections of antimon. et sod. tartras. The subcutaneous nodules should be excised.

### Dracontiasis

#### (Guinea-worm Disease)

Etiology. Dracontiasis is caused by the nematode *Dracunculus medinensis*. The adult worms are found in man. They measure from 6 inches to 3 feet long and about  $\frac{3}{16}$  inch in diameter. The embryo worms are present in a small crustacean (*Cyclops*), and man is infected through drinking contaminated water. The worms are liberated in the stomach, enter the intestine, and finally pass to the subcutaneous tissues, increasing in size. The adult female worms penetrate the skin, usually on the arms or legs, and the uterus discharges embryos on to the surface of the skin for about 3 weeks. If the skin is in contact with water, as is usually the case, embryos pass into the water and then again into the *Cyclops*. The adult worm then dies, and may be discharged from the skin or become calcified, or an abscess may form around it. Locality: Dracontiasis is met with in India, in Egypt near the Nile, in West Africa, Uganda and the East Indies.

Clinical Findings. During the invasion period, before the adult

worm has matured, there may be constitutional disturbance with fever, nausea, vomiting and eosinophilia. As the worm works its way to the surface urticaria may develop. An itching or burning spot may then be noted at which a blister forms. This ruptures and the head of the worm may be seen below. The embryos are discharged, as described above.

*On Examination:* The adult worm can usually be seen or felt beneath the skin.

*Treatment. Prophylactic.* Water used for drinking or for washing food utensils should be sterilised by boiling.

*Curative.* The worm must not be extracted until it has discharged all its embryos, as rupture of the worm may lead to severe toxic disturbances or to secondary infection resulting in cellulitis. A few drops of cold water on the skin near the orifice cause the worm to discharge its embryos. After about 3 weeks, when no more embryos are produced by application of cold water, the worm may be removed by traction.

An alternative method of treatment consists in cooling the skin with an ethyl chloride spray. The worm contracts and becomes prominent. Several incisions are then made transversely across it. The worm is hooked up through these incisions and divided, the pieces pulled out and the sinuses disinfected with 1 in 30 carbolic acid.

## CHAPTER XIV

### DISEASES DUE TO PHYSICAL AGENTS

#### Caisson Disease

(*Compressed Air Illness. Diver's Palsy*)

**Definition.** A disease due to exposure to compressed air.

**Etiology.** This is a disease of workers in caissons, who are exposed to compressed air when employed as divers or as builders of bridges, tunnels or skyscrapers. There is usually no risk unless the pressure is increased by more than that of one atmosphere (+ 15 lb. per square inch). The danger is intensified by the length of exposure to the increased pressure. Thus divers are less often affected, because, although when they go to a great depth the pressure in their helmets is high, they work at such a pressure for only short periods. The symptoms appear on decompression on return to the surface.

**Pathology.** The increased pressure in the caisson causes excess of oxygen and nitrogen to be taken up by the blood. The oxygen combines with the tissues and does no harm. The nitrogen is also absorbed by the tissues, especially by fat and by the central nervous system, and after a time the blood is completely saturated with nitrogen. If the pressure is now rapidly reduced, as the worker comes to the surface, the nitrogen is liberated from the tissues in the form of bubbles; these bubbles may appear in the fatty tissues, spinal cord, brain, intestines, and also in the blood. Thus the capillaries may be obstructed by gas emboli or the heart filled with gas. Permanent damage may be done to tissues by thus cutting off their blood supply. Fat people absorb more nitrogen and so are more prone to the disease. Post-mortem: Numerous gas bubbles may be seen in the brain, the cord, especially in the lower thoracic portion, in the subcutaneous tissues, the heart, and in certain viscera, such as the liver.

**Clinical Findings.** The patient usually gives a history of a rapid return to the surface from a depth, and notices symptoms about  $\frac{1}{2}$  to 3 hours later. In mild cases there is headache and pains in the joints, especially the knees and ankles, and in muscles (the "bends"). The legs are usually affected and the joints are kept flexed. In more severe instances there is giddiness (the "staggers"), abdominal pain, nausea, vomiting, shortness of breath (the "chokes"), intense itching of the skin (the "itch"), and paralysis, usually paraplegia, or collapse and death may rapidly ensue.

**Differential Diagnosis.** The history of the case and clinical picture render the diagnosis obvious. In some cases the patient is first seen in coma, and in others the condition may suggest an acute abdominal lesion.

**Course and Complications.** Unless adequate treatment is given at once, permanent nervous damage or death may ensue. Secondary



hæmorrhage may occur in the affected tissues. Chronic arthritis and aseptic bone necrosis may develop as complications.

**Prognosis** This depends entirely upon the severity of the condition and the means available for early treatment.

**Treatment** *Prophylactic* Fat men, and those suffering from diseases of the heart, lungs or kidneys, or men addicted to alcohol must not be employed as caisson workers. Inflammation of the Eustachian tubes is also a contra indication, as there is a risk of rupture of the tympanic membrane owing to inability to equalise the pressure in the middle ear by swallowing movements during the time the external pressure is being raised. Men should not work longer than a 1 hour shift at a pressure of + 50 lb., longer shifts are permissible for lower pressures. When working at increased pressures decompression must be gradual. The men pass through a series of air locked chambers, where the pressures are gradually lowered a due stay being enforced in each chamber. Exercise and the inhalation of oxygen are also important during the decompression.

*Curative* The patient should be placed in a chamber ("medical air lock") and the pressure raised to that at which he was working. After half an hour he is very gradually decompressed. If no chamber is available, the patient may be lowered to the pressure at which he was originally working, and gradually brought to the surface. If this is not feasible oxygen and morphine should be administered.

### Mountain Sickness

#### (Anoxæmia)

**Definition** Illness resulting from exposure to a low barometric pressure.

**Etiology** Mountain sickness results from ascents to great heights, usually over 12,000 feet, as in climbing or flying.

**Pathology** There is a deficient supply of oxygen to the blood. Compensatory changes may occur, such as increase in the number of red cells in the blood (erythrocytosis), there are no nucleated red cells, but the reticulocytes are more numerous. There is alkalæmia with a fall in the alkali reserve.

**Clinical Findings** In rapid flying ascents to over 25,000 feet death may occur, after a preliminary stage of dulling of all the mental faculties, followed by muscular paralysis. In more gradual ascents the patient complains of headache, muscular weakness, giddiness, palpitations, dyspnoea, nausea, vomiting and fainting attacks. He appears cyanosed.

**Treatment** A portable oxygen apparatus should be used at high altitudes.

### Sea-Sickness

#### (including Train, Car and Air Sickness)

**Definition** Sickness occurring on the sea, in a train, a car, or an aeroplane.

**Etiology.** There are various theories as to the causation of these varieties of sickness, such as: Labyrinthine, ocular or splanchnic disturbances; a neurosis, resulting from auto-suggestion; acidosis and hypoglycæmia. Train, car and air sickness are probably closely allied conditions. *Predisposing causes:* 1. Age: Infants and the aged are immune, all other ages are susceptible. 2. Those subject to migraine or suffering from diabetes mellitus or nephritis, and pregnant women are especially liable.

**Pathology.** It has been shown that before vomiting occurs there is an increased output of ammonia in the urine, with acetone bodies. There is also a preliminary hyperglycæmia followed by hypoglycæmia. Acidosis is more severe when the vomiting stage is reached.

**Clinical Findings.** The unfortunate victim feels squeamish, with a tendency to yawn and salivate. He may have headache and disturbance of vision, such as diplopia. The vomiting is usually ushered in by more profuse salivation; in severe cases there is marked collapse, the sufferer loses interest in life and wishes for a speedy end to his torments. The face is pale or greenish, the skin is cold, the pulse rapid and blood pressure low. The output of urine is diminished and diarrhœa or constipation may occur.

**Differential Diagnosis.** It is important not to overlook any abdominal condition, such as acute appendicitis, intestinal obstruction or a perforated gastric ulcer. Attention must be paid to the presence of abdominal rigidity in organic abdominal lesions and alterations in temperature and pulse rate recorded on an hourly chart. The result of enemas is of diagnostic value in intestinal obstruction.

**Course and Complications.** Some individuals are never able to overcome sea-sickness; in others accommodation is rapidly established. Concentration of the urine may lead to bladder irritability, resembling that due to cystitis.

**Prognosis.** Sea-sickness is never fatal; pregnant women do not miscarry.

**Treatment.** A meal rich in carbohydrate should be taken before the voyage, and the blood sugar kept up subsequently by eating lump sugar at the earliest appearance of symptoms. Fats should be avoided. Sedatives, such as Chloretone (chlorbutol B.P.) gr. 5 in a cachet, may be taken twice a day. The sufferer should keep warm, have plenty of air and lie down.

### Heat-stroke

(Sunstroke)

**Definition.** Illness resulting from exposure to the sun.

**Etiology.** Heat-stroke usually results from exposure to the sun. It generally occurs in the tropics, when the shade temperature reaches 110° F. White races are very susceptible. *Predisposing causes:* Debility from other diseases, alcoholism and constipation.

**Pathology.** The heat-regulating mechanism is disturbed. It is not known whether toxins are formed in heat-stroke. Post-mortem, the right heart is dilated.

**Clinical Findings** The patient gives a history of exposure to sunlight. He is suddenly taken ill with headache and may vomit and have diarrhoea. The patient collapses and may rapidly become unconscious.

**On Examination** The face is flushed, the skin is hot and dry, the pupils are dilated, the pulse and respirations are rapid and the temperature is raised to 109°F or higher. Convulsions may occur. The knee jerks are absent. The breathing may become irregular, of the Cheyne-Stokes type, and the pupils are constricted before death.

**Differential Diagnosis** A blood film should be taken to exclude malaria.

**Course and Complications** Death may rapidly ensue. Complications include dilatation of the right heart and oedema of the lungs. Cerebration may be subsequently impaired if the patient recovers and neuritis may be troublesome.

**Treatment Prophylactic** The bowels should be kept open daily, plenty of fluid consumed but alcohol should not be taken to excess. The head, neck and spinal cord should be adequately protected from the sun.

**Curative** The temperature is lowered by applying ice to the head, neck and spine, spraying the body with ice cold water, and, if necessary, by giving a rectal injection of ice cold water. When the temperature falls to 102°F these measures must be discontinued. Venesection should be performed if the patient is cyanosed. An intravenous injection of quinin dihydrochlor gr 10 in 20 mls of distilled water should be given slowly if there is any doubt as to the condition being due to malaria.

### Heat Exhaustion and Heat Cramp

**Definition** Illness resulting from exposure to heat.

**Etiology** Heat exhaustion is prone to develop in hot moist atmospheres when evaporation from the skin is low. Heat cramp results from exposure to dry heat, the excessive sweating leading to diminution of the blood chlorides. Stokers, miners and iron and steel workers are subject to heat exhaustion without any direct exposure to sunlight.

**Clinical Findings** The patient has usually been working in a very hot atmosphere such as that which a stoker must endure. He becomes weak, giddy, and may sweat and collapse. In some instances there is vomiting and diarrhoea, or painful cramps may be felt in the legs.

**On Examination** The patient is usually pale, the skin is moist and the temperature may be normal, subnormal or raised to about 100° or 101°F.

**Treatment** The patient should be moved to a cool place, and stimulants such as an ether and ammonia mixture given by mouth (Ammon carb gr 1, liq ammon fort m 1, æther m 6, aq menth pip dest ad fl oz 1). An enema should be given if there is constipation. If the temperature is subnormal the patient should be placed in a bed and warmed with hot bottles. Heat cramp may be prevented or

relieved by drinking 0.1% sod. chlorid. solution, or by taking by mouth tablets of sod. chlorid., gr. 15, up to 15 or 20 in the day.

### Frost-Bite

**Definition.** The harmful effects produced by cold on peripheral parts of the body.

**Etiology.** Frost-bite results from exposure to severe cold, especially if the individual is in a high wind or is at a great altitude. Trench foot is a variety of frost-bite. The causative factors here are cold, not necessarily of a severe degree, stagnation of circulation due to standing in wet and muddy trenches, ill-fitting boots and tight clothing on the legs.

**Pathology.** Severe cold produces peripheral cutaneous vasoconstriction, followed by liberation of H-substance, vasodilatation, damage to vessels and transudation of fluid into the surrounding tissues. This results in œdema and bulla formation. In more severe cases of frost-bite thrombosis and gangrene ensue. The effect of high altitude and oxygen-lack is an increase of pulse rate up to 180 to 250 per minute, with a reduction in the output (minute-volume) of blood from the heart.

**Clinical Findings.** Frost-bite may occur very rapidly, e.g., if a metallic object is touched by the tip of a bare finger in severe cold or at a high altitude. In other cases, where the exposure is more prolonged, as may happen when tramps sleep out in the winter, there is first a feeling of burning in the extremities followed by a lack of all sensation. The onset of trench foot is much more insidious, taking often days or weeks.

**On Examination:** Various stages of frost-bite may be seen, such as a white and waxy appearance of the skin in mild cases. In more severe ones the skin is red or almost black, with bulla formation and gangrene. Trench-feet are usually bluish-red. Varying degrees of hypoaesthesia to light touch, pin-prick and temperature sensation are present in frost-bite and trench foot.

**Course and Complications.** Pain is experienced in the extremities as recovery takes place. The nails may be shed or portions of the extremities lost by gangrene.

**Treatment.** *Prophylactic.* Protection by means of adequate clothing, dry gloves and ear-flaps, is important. Properly fitting supple, well-oiled boots must be supplied to soldiers in trenches. Puttees and darned socks should not be worn and socks should be changed when wet as soon as feasible. The body temperature should be kept as warm as possible.

**Curative.** If a foot or hand becomes numb or the nose goes white, the affected part should be warmed by taking off the boot or glove and placing the limb between the thighs or under the arm of a comrade. The nose should be warmed between the hands. In more advanced cases local application of heat in any form must be avoided, and friction, which includes rubbing with snow, must, on no account, be applied to the affected parts. These should be cleaned gently, using gauze soaked

in 1/1,000 proflavine then carefully dried and covered with sterile gauze and several layers of wool. Bullæ which have burst or gangrenous areas should also be treated with 1/1 000 proflavine. General treatment consists in combating shock by the administration of hot drinks. If the body temperature is subnormal the bed should be warmed with hot bottles which must not be placed near the affected parts. Prophylactic injections of antitetanic serum should be given (see p 58.) In cases developing at high altitudes the administration of oxygen is of great value. Amputation may be required for gangrene but this should be delayed as long as possible, as partial recovery is likely to ensue.

### Electric Shock and Burns

**Definition** The harmful effects produced by electric currents

**Etiology** Electric shock is usually accidental, as by contact with a live wire on an electric railway or tramway system, by touching an electric heater when in a bathroom, or by contact with a house lighting system. Death in the bathroom is due to a fault in the electric system whereby some of the current is entering the switch. The victim is usually in the bath, which acts as a condenser, and on switching off the heater his body is subjected to a high current. The result is not usually fatal with voltages under 300 for the direct current or under 100 with an alternating current. Very high voltages of alternating current, on the other hand may do little or no harm. Lightning may cause electric shock, and burns may occur from X rays or diathermy used in medical treatment.

**Pathology** The body is often charred locally, the blood is fluid and small hæmorrhages may be found in the brain. The heart may be arrested by ventricular fibrillation, this causes death in 90% of cases. In death from lightning an effusion is found under the scalp without any superficial abrasion. Burns of varying degree are seen on the body.

**Clinical Findings** The patient is often killed outright when struck by lightning the clothes may be stripped off the body. In other cases the victim is rendered unconscious but slowly recovers experiencing great pain as the circulation is restored. In slighter degrees of electric shock the patient, who has gripped a live wire is unable to relax his hold is terrified and feels severe pain. Electrical burns of all kinds are very slow in healing. Albuminuria may be noted subsequently due to the presence in the blood of abnormal protein substances.

**Treatment** The current should be switched off and the patient removed. If the current cannot be cut off, the hands of the rescuer should be protected by rubber gloves or some dry, thick material. He should also stand on a dry substance, such as bricks or cloth. Artificial respiration should be applied for several hours if the patient is unconscious.

## CHAPTER XV

### THE POISONS

*Introductory.* Marriott has helped to simplify the treatment of acute poisoning by concentrating on the basic principles. These are concerned with the relief of asphyxia, stimulation of the respiratory centres, an adequate supply of oxygen, gastric lavage, neutralisation of

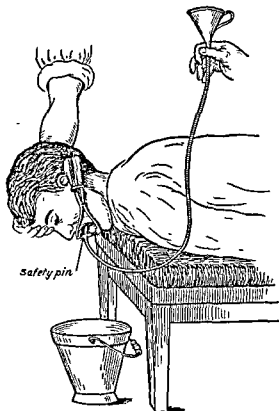


FIG. 71. MARRIOTT'S METHOD OF GASTRIC LAVAGE IN CASES OF POISONING.

corrosives, treatment of coma, relief of dehydration, replacement of chlorides, assuaging pain and calming delirium or convulsions.

Acute poisoning is usually due to substances taken by inhalation or by mouth, rarely by injection. Carbon monoxide is the most common cause of fatal poisoning in England and in America, and the treatment by artificial respiration and the inhalation of carbon dioxide and oxygen is considered on p. 747. To rid the body of swallowed poisons, which have not yet been absorbed, gastric lavage is of the greatest value. Emetics should only be given if the requisites for gastric lavage

are not available. Purges are seldom required. Gastric lavage is contra-indicated if strong corrosives, such as strong nitric, sulphuric or hydrochloric acid, caustic potash or soda, or strong ammonia have been swallowed. In such cases the corrosive should be neutralised. Strong acids can be neutralised with four tablespoonfuls of light or heavy magnesia to a pint of water, half a pint being used for a child. To neutralise strong alkalis six tablespoonfuls of vinegar or the juice of six lemons are added to a pint of water. Apart from poisoning by such strong corrosives, it is always advisable to wash out the stomach, even if the patient has vomited, or if several hours have passed since the poison was taken. Gastric lavage can be safely used in the case of poisoning by oxalic or carbolic acid, or by lysol.

Very thorough lavage should be given, using 2 gallons of warm water. Great care should be taken to prevent the fluid from regurgitating into the lungs, and for this purpose the method recommended by Marriott should be followed. The patient is placed prone with his head hanging over the end of the couch or bed, the forehead being supported by an assistant (see Fig 71). A Jaques' firm rubber stomach tube (œsophageal tube), size 23-30 English catheter gauge, 60 inches long is used for an adult. A safety pin is passed through the wall, not the lumen, of the tube, 20 inches from the end which will lie in the stomach. Any false teeth having been removed and the mouth being kept open by a gag, the lubricated tube is passed into the mouth until it reaches the pharyngeal wall, and is then pushed quickly down into the stomach. The safety pin in the tube should lie about  $\frac{1}{2}$  inch outside the incisor teeth. The tube is passed with the patient in the prone position, the operator sitting or kneeling on the floor to do so. A funnel is attached to the upper end of the tube and a pint of warm water run into the stomach. The funnel is then inverted over a pail on the floor and the stomach contents are syphoned out. In this way 2 gallons of water are used for the lavage. Care must be taken that the tube does not alter its position while the lavage is being carried out. If emetics are required a subcutaneous injection of apomorphin hydrochlor gr 1/10 will produce vomiting in 5 minutes. Other emetics which may be used include pulv ipecac gr 30, mustard or salt  $\frac{1}{2}$  oz in a tumbler of warm water. Dehydration and loss of chlorides are liable to occur if there has been severe vomiting and diarrhoea. If the patient can swallow he should be given half normal saline (NaCl gr 40 to a pint) sweetened with dextrose 1 oz and the juice of two oranges. If he cannot swallow, normal saline should be given by drip transfusion, either rectally or intravenously. Sufficient fluid should be administered to relieve thirst, improve the blood pressure and increase the urinary output, but care must be taken not to give sufficient to embarrass the heart or cause œdema of the lungs.

### Mercury Poisoning

**Etiology** Mercurial poisoning may occur 1 As an occupational disease, in miners of mercury, thermometer, barometer or looking glass makers, and felt hat makers who use mercury nitrate 2 If mercury

(usually the perchloride) is taken for suicidal or homicidal reasons. 3. In accidental contamination of foods or by accidentally drinking perchloride of mercury. 4. In the therapeutic use of mercury, due to gradual overdosage or personal idiosyncrasy, as in the treatment of syphilis.

**Pathology.** *In acute poisoning there is found post-mortem intense inflammation of the mucous membrane of the stomach and intestines. The stomach mucous membrane may be greyish white, and a greyish membrane may form in the large intestine. The kidneys show the changes of acute nephrosis (see p. 451).*

**Clinical Findings.** *Acute Poisoning.* This usually results from taking perchloride of mercury solution by mouth, either accidentally or suicidally, or by using it as a vaginal douche. Within a few minutes of ingestion there are severe epigastric pains and vomiting. The vomit may contain blood. Diarrhoea rapidly follows and the stools also may be bloodstained. Prostration and collapse follow according to the severity of the intoxication. The blood : This may show an increase of urea and non-protein nitrogen. The urine : This is highly acid, and may contain albumin, blood and casts. Suppression of urine may ensue (see p. 451).

**Chronic Poisoning.** Therapeutic overdosage with mercury is characterised by salivation, a metallic taste, stomatitis, gingivitis, offensive breath and diarrhoea. In industrial poisoning there may be also headache, lassitude, anæmia, loosening of teeth, albuminuria, a rise of blood pressure and marked tremors of an intentional variety. Mercurial dermatitis, with erythema and desquamation, may appear. Mercurial erythism is sometimes noted, and is characterised by excitability, shyness with strangers, insomnia, depression and giddiness.

**Differential Diagnosis.** The diagnosis is usually apparent from the history and typical clinical findings. Mercury may be present in the faeces.

**Course and Complications.** The course depends upon the amount of mercury ingested. Complications include nephritis, anæmia and colitis.

**Prognosis.** This is very grave in acute cases, but has been improved by modern treatment. Death may occur in a few hours or days. In chronic cases recovery usually results, and depends upon the rapidity of the recognition of intoxication and withdrawal of the poison. Tremors may persist for several years.

**Treatment.** *Acute Poisoning.* The stomach should be washed out with 2 gallons of a warm saturated solution of sodium bicarbonate, and 8 oz. of a saturated solution of magnesium sulphate left in the stomach. A soap and water enema should then be given. 10 oz. of a 4% solution of sodium bicarbonate (gr. 180 in 10 oz.) are injected intravenously, and this is repeated again next day. The patient should drink : Acid. pot. tart. gr. 10, sod. cit. gr. 30, syr. aurant. m. 20, aq. ad fl. oz. 8. Fl. oz. 8 four-hourly.

The essential feature is to render the urine alkaline and keep it so.

**Chronic Poisoning. Prophylactic.** Workers in a dangerous occupa-



tion should be subjected to periodical medical examinations. When mercury is being administered a watch should be kept constantly for early signs of mercurialism.

**Curative.** The mercury administration must cease, or the worker be removed from exposure to the poison. Elimination is aided by administering mag sulph gr 60 to 120 daily. For mercurial dermatitis a lotion of 1% sodium hyposulphite should be applied (gr 4 to fl oz 1).

### Lead Poisoning

#### (Plumbism Saturnism)

**Etiology.** Lead may enter the body through the alimentary tract, it may be inhaled as dust, or absorbed through abrasions in the skin or mucous membranes. Lead poisoning is chiefly of importance as an occupational disease. The following are the chief causes. 1 *Occupations* Workers in red and white lead, painters, potters, brass foundrymen, miners, rubber mixers, accumulator manufacturers and printers. 2 *Food* Soft water may be contaminated by lead pipes, beer may be similarly affected, cider may be tainted by lead glaze in jars, and wine by subacetate of lead added as a sweetening agent. Tinned foods may cause poisoning, especially oily fish in soldered tins and illness has resulted from eating cakes coloured yellow with lead chromate. 3 *Cosmetics* Hair dyes, toilet powders or face creams may contain lead. 4 *Drugs* Ointments containing lead may cause poisoning when applied to broken surfaces, or lead lotions used as an eye or vaginal douche. Lead pills, made from diachylon plaster, were formerly used to obtain abortion, but the sale has been checked by the Poisons Act. Lead tetraethyl can be absorbed through the skin. **Predisposing causes.** Women and children are more susceptible than men, and negroes than white men. Debility from any acute illness and chronic alcoholism are also predisposing factors.

**Pathology.** Lead absorbed from the alimentary canal is carried to the liver and may then pass into the systemic circulation or be excreted in the bile. It is conveyed by the blood stream to all the tissues of the body, but is stored especially in the bones. It circulates in the blood plasma as a colloidal phosphate and accumulates in the solid parts of the bones, probably as an insoluble triple phosphate,  $Pb_3(PO_4)_2$ . It may be liberated from the bones and flood the blood. It is excreted chiefly in the faeces, to a slight degree in the urine, and less still by the skin. Inhaled lead dust is carried direct to the systemic circulation. A slight increase of the acidity or alkalinity of the blood appears to result in liberation of lead from the bones, as does also any agent which causes solution of calcium salts from the bones. Post mortem Gastro-enteritis is found in acute lead poisoning, in chronic cases nervous lesions such as degeneration of anterior horn cells, peripheral neuritis, and muscular atrophy may be found. The amount of lead present in the skeleton varies between 0.2 and 0.8 G. Such lesions as arteriosclerosis and chronic nephritis are not indubitably due to lead.

**Clinical Findings.** *Acute Lead Poisoning.* This may be a primary disease, caused by a large dose of lead taken with suicidal intent, or

accidentally, as in cakes coloured with lead chromate. If a large dose of lead is swallowed the patient suffers from burning in the mouth, thirst, dysphagia, intestinal colic, vomiting, cramps in the legs and convulsions. The bowels are constipated. Acute symptoms or "toxic episodes" may also occur during the course of chronic lead poisoning, and these are described later.

*Chronic Lead Poisoning.* The worker complains of lassitude and dyspnoea on exertion. He becomes constipated, loses his power of concentration, has vague pains in the arms and shoulders and loses weight. Cramps may occur in the legs. Periodically acute symptoms, known as "toxic episodes," may occur. Thus the subject of chronic plumbism may have severe intestinal colic. This usually follows a period of marked constipation; the pain is generally hypogastric, and may last for several days. Cramp may also be acute in the legs, bladder or uterus, with menorrhagia or abortion if the patient is pregnant. Acute nervous symptoms such as mania or convulsions, coma or delirium may develop, due to a meningo-encephalopathy. Other nervous lesions may ensue more insidiously, such as optic neuritis and optic atrophy with resulting blindness, or the worker may experience difficulty in using the muscles especially concerned with the performance of his duties.

*On Examination:* The patient is pale and the skin has a greyish tinge. The teeth are often carious and pyorrhoea is present. A blue line may be seen on the gums adjacent to carious teeth or on the mucous membrane of the cheek or lips opposite the bad teeth. This line consists of a series of blue-black dots, best seen with a hand lens, situated just below the margin of the gums and in the subepithelial tissue. It is due to lead sulphide, the  $H_2S$  being formed by putrefaction in the mouth. Tremors of the tongue and hands may be noted. Peripheral nerve lesions are generally bilateral. They include: Antebrachial paralysis, with wrist drop, due to paralysis of the posterior interosseous branch of the musculo-spiral nerve; the supinator longus muscle is therefore spared. Aran-Duchenne paralysis, with wasting of the small muscles of the hand and thenar and hypothenar eminences, a "claw hand" resulting. Brachial paralysis, the biceps, supinator longus, brachialis anticus and deltoid muscles are affected. Peroneal paralysis, with foot drop and paralysis of the peroneal muscles, extensor longus digitorum and extensor proprius hallucis. Recurrent laryngeal nerve paralysis, with adductor cord paralysis, may occur. In some instances there is a diffuse muscular paralysis and in others a cerebellar ataxia appears. Sensory changes are generally absent. The reaction of degeneration is obtained in the affected nerves.

*Examination of a Patient during an Attack of Colic:* The pain is obviously very severe, the patient is pale, sweating and holds his hand to his abdomen, as pressure affords some relief; the abdominal wall is not really rigid and is hollow rather than distended. The temperature is subnormal and the pulse slow. The blood: There is an anaemia. The red cells show "stippling" (punctate basophilia) and at least 100 red cells per million red cells must be so stippled to be diagnostic. These

stippled cells are reticulocytes. The white cells are usually normal. The faeces. These usually contain lead, as does the urine if lead has reached the blood stream. The cerebro-spinal fluid. In the convulsive stage it is under pressure and contains an excess of lymphocytes.

**Differential Diagnosis.** It should be remembered that the symptoms of plumbism may develop after an individual has ceased to be exposed to lead. Various factors may cause lead to be liberated from the bones into the blood. The diagnostic features are the blue line, the punctate basophilia and the presence of lead in the urine. As traces of lead may be found in the urine in health, due to lead eaten with food at least 0.1 to 0.3 mg lead per litre of urine must be present to be diagnostic.

**Course and Complications.** The course is characterised by the "toxic episodes" described above. Complications include such conditions as arteriosclerosis, chronic nephritis and gout.

**Prognosis.** Recovery is apt to be slow. Colic usually rapidly responds to appropriate treatment, paralyses tend to persist unless exposure to lead is stopped at the earliest symptom, recovery occurring first in the muscles last affected. Death is especially liable to occur in maniacal cases.

**Treatment.** *Prophylactic.* Measures should be taken to prevent workmen inhaling lead dust, these include the use of wet processes and the employment of adequate fans and respirators. Workers in lead should wash their hands before eating and change their clothes on leaving work. Periodical medical examinations at least monthly should be held to detect early signs of plumbism, and any suspicious cases suspended from work and kept under observation. A high calcium diet, such as one containing one to two pints of milk daily should be taken.

*Curative.* In acute poisoning accidental or suicidal the stomach should be emptied by an emetic such as mustard,  $\frac{1}{2}$  oz in  $\frac{1}{2}$  pint of warm water, or preferably washed out with 2 gallons of warm water containing 2 oz of mag sulph and a saline aperient is then given, such as Mag sulph, sod sulph aa gr 240 acid sulph dil m 30, aqua ad fl oz 10. Fl oz 10 to be taken every 4 hours until the bowels are thoroughly evacuated. For abdominal colic hot flannels should be applied locally, milk should be drunk and calcium lactate gr 30 taken by mouth t i d. When the pain is very severe an intravenous injection should be given slowly of 15 mls of calcium gluconate (B.P. Add). A subcutaneous injection of atropin sulph gr  $\frac{1}{60}$  and morphin sulph gr  $\frac{1}{4}$  is also useful for relieving the pain of severe colic. In the acute exacerbations of chronic lead poisoning efforts are directed to retain lead in the bones by giving a diet rich in calcium such as milk 4 pints in 24 hours together with calcium lactate gr 30 t d s by mouth.

"Deleading" measures must only be instituted when the acute symptoms have subsided. This is accomplished by establishing a negative calcium balance. A calcium poor diet is given, such as meat, potatoes, bananas, apples, rice, tomatoes etc. No milk, eggs or green vegetables should be given. Ammon chlorid gr 15 is given in a glass of water four hourly. This is continued for 2 or 3 weeks, as long as

there is no nausea and headache, the patient being kept on a calcium poor diet. If toxic lead symptoms occur the treatment is discontinued and a high calcium diet given. The ammonium chloride produces its effect by the acidosis it provokes. Potassium iodide was formerly used; this does increase the excretion of lead in the fæces, but it has now been largely replaced by the ammonium chloride treatment. The resulting anæmia should be treated with an iron tonic or by blood transfusion. After the patient has been cured he should not be allowed to return to work in which he is exposed to lead.

### Arsenic Poisoning

**Etiology.** Arsenical poisoning may be due to: 1. Occupations, such as extracting white arsenic from arsenical pyrites or preparing sheep-dip. Arsine may be inhaled in chemical works or in submarines, where it is liberated from battery plates. 2. Administration of arsenic with homicidal or suicidal intent. Weed-killers, fly-papers or rat pastes may be used. 3. Food-stuffs accidentally contaminated, such as glucose in the preparation of beer, the sugar covering sweets, and the skin of apples which have been sprayed with an arsenic solution as a parasiticide. 4. Therapeutic administration of arsenic, either due to an overdose being given or to idiosyncrasy of the patient. It may occur with such preparations as Fowler's solution, sodium cacodylate, Atoxyl (sod. aminarsonas B.P.C.), tryparsamide or neoarsphenamine (see p. 572).

**Pathology.** In acute cases there is inflammation of the mucous membrane of the stomach and upper part of the small intestine, and cloudy swelling may be found in the liver, heart and kidneys. In cases of chronic poisoning fatty degeneration is present in most of the organs of the body. Arsenic can be detected in the body for many months after death.

**Clinical Findings. Acute Poisoning.** Directly after taking a poisonous dose the patient experiences dryness in the throat, and this is soon followed by a burning sensation in the epigastrium. He feels sick and vomits. Intestinal colic and severe diarrhoea usually follow and there may be cramps in the legs. Small amounts of blood may be present in the vomit or the stools.

**On Examination:** In a severe case the patient is cold and collapsed, the pulse rapid and of small volume. The acute symptoms following poisoning by the neoarsphenamine preparations are described on p. 572.

**Chronic Poisoning.** The symptoms here are insidious. In the early stages there is irritation of the nasal and conjunctival mucous membranes and later of the pharynx and larynx. If arsenic is being given as medicine these symptoms should be a sufficient warning of an overdosage. Further administration of arsenic leads to abdominal discomfort, loss of appetite, nausea, vomiting, tetany, intestinal colic and diarrhoea. The tongue has a silvery white fur. There is a generalised pigmentation of the skin, especially in the flexures. Keratosis may develop on the soles and palms. Arsenical neuritis causes pains in the

legs and arms, cramps and paresis of the legs Herpes may develop Sudden blindness due to optic atrophy may occur with Atoxyl (sod aminarsonas B P C), or tryparsamide Arsenic may be found in the urine, faeces and vomit and also in the nails or hair

**Differential Diagnosis** In general practice it is a delicate matter to reveal one's suspicions of arsenic poisoning, but it is the duty of the doctor to have the urine, faeces, nails or hair examined in any doubtful case Acute arsenical poisoning may be mistaken for cholera or other forms of acute gastro enteritis Chronic arsenical poisoning may be mistaken for gastro-enteritis, colitis, carcinoma of the stomach or Addison's disease

**Course and Complications** These depend on whether or not arsenic is continuously administered In progressive cases death usually occurs from heart failure with ascites and oedema

**Prognosis** If fatal, death from acute arsenical poisoning usually occurs in a few hours or days The prognosis in chronic cases depends upon the recognition of the disease and the cessation of the administration of arsenic

**Treatment** *Acute Poisoning* The stomach should be emptied by giving an emetic, such as mustard ( $\frac{1}{2}$  oz in  $\frac{1}{2}$  pint of warm water) until vomiting occurs or preferably by gastric lavage Freshly prepared ferric hydroxide should be used in gastric lavage, as this forms an insoluble arsenite It is made by adding sod bicarb to liq ferri perchlor fl oz 2 until effervescence ceases The precipitate of ferric hydroxide is filtered off and added to 2 gallons of warm water An aperient should be given after an hour consisting of mag sulph gr 240 in half a tumbler of water General treatment consists in keeping the patient warm, administering stimulants such as digitalin gr 1/100 hypodermically or strophanthin gr 1/250 intravenously, and if the abdominal pain is very severe a hypodermic injection of morphin sulph gr 1/6 to 1/4 may be given

In chronic poisoning after discontinuation of the arsenic, the treatment is symptomatic for the debility and neuritis The treatment for poisoning by neocarsphenamine is considered on p 572

### Alcoholic Poisoning

**Etiology** Alcohol in any form may cause symptoms of poisoning It is usually due to ethyl alcohol, less frequently to methyl alcohol

**Predisposing causes** 1 Occupation Public house keepers, barmen and commercial travellers. 2 Heredity A neuropathic predisposition 3 Worry and pain The habit may persist after the drug has been ordered by a doctor during illness

### Acute Alcoholic Poisoning

#### (Physiological Inebriation)

**Pathology** Alcohol is rapidly absorbed from the stomach and intestines and passes to the blood and cerebro spinal fluid Post-mortem there may be hyperaemia of the gastric mucous membrane and of the brain

**Clinical Findings.** The patient suffering from acute alcoholic poisoning is drunk, but the definition of drunkenness, from the medico-legal aspect, has not been satisfactorily established. A person is usually considered to be drunk when, as a result of taking alcohol, he is not able to perform with his normal skill or facility the ordinary actions of his life. The higher centres are first inhibited and then the lower. In the early stages there is diminution of cerebral restraint. The individual loses any sense of shyness and may become talkative or emotional. The onset of muscle fatigue is delayed. The finer acts of co-ordination are disturbed, and later there is unsteadiness of gait, inability to walk, and finally coma. In some instances there is no excited stage, but marked depression from the onset.

**On Examination :** The breath smells of alcohol, the face is usually flushed but may be pale, the skin is moist, the pupils dilated and the pulse rapid. There is difficulty in the pronunciation of such words as "British Constitution," and inability to walk steadily along a line. In alcoholic coma the pupils are dilated, the respiration is slow, the pulse feeble, the body temperature subnormal, the limbs flaccid, the deep reflexes diminished and the plantar response is flexor. The urine : The amount of alcohol present in the urine has been suggested as a medico-legal test for drunkenness. It is said that no man whose urine contains 150 mg. alcohol per 100 c.c. is fit to drive a car. The alcohol in the urine bears a direct relationship to the alcohol content of the blood.

**Differential Diagnosis.** Drunkenness must be distinguished from : Excitement due to other causes. Coma due to other causes, such as hypoglycæmia after a dose of insulin. The presence of disease or injury in a person who has taken a small amount of alcohol.

**Course and Complications.** In the majority of cases the patient recovers after a night's rest, but alcoholic coma and pulmonary œdema are always causes of great anxiety. Nephritis or pneumonia may ensue when the patient is exposed to cold, of which he is unaware.

**Prognosis.** This is usually good, but death may occur in coma from respiratory or cardiac failure.

**Treatment.** In the majority of cases no treatment is required beyond an aperient, a night's rest in bed and aspirin gr. 10 to 15 to relieve the subsequent headache. In severe cases the patient should be given an emetic ( $\frac{1}{2}$  oz. mustard in  $\frac{1}{2}$  pint of warm water), or preferably gastric lavage should be performed with 2 gallons of warm water containing sodium bicarbonate (1 oz. to a pint), and a pint of hot coffee left in the stomach. Collapse may be further treated by an electric cradle, hot bottles and stimulants such as Coramine (nikethamidum B.P.Add.) 1.5 ml., or strychnin. hydrochlor. gr. 1/60 hypodermically four-hourly. If the patient is comatose inhalation of 10% CO<sub>2</sub> in 90% oxygen helps to wash out the alcohol and to oxidise it. This treatment usually restores the patient to consciousness in half an hour.

### Chronic Alcoholic Poisoning

**Pathology.** Post-mortem there may be fatty infiltration and degeneration of the heart, atheroma of the aorta, fatty infiltration and

cirrhosis of the liver, dilatation of the œsophageal veins, atrophy of the gastric mucous membrane, chronic nephritis of varying types, congestion of the meninges, excess of fluid in the pia arachnoid space ("wet brain"), degeneration of cortical motor cells and peripheral neuritis

**Clinical Findings** The effects of chronic alcoholism may be considered under the systems chiefly involved

*The Alimentary System* The patient may complain that the appetite is poor, and there may be morning nausea or vomiting, the stools being rather loose. *Hæmatemesis may occur or bleeding from the rectum due to piles*

*On Examination* The patient is often somewhat obese, dilated venules are seen on the face, the conjunctivæ are watery and injected, the tongue is furred and tremulous the throat congested and the breath "heavy". The liver may be enlarged and ascites present

*The Nervous System* Peripheral neuritis may be the chief complaint. The patient is often a woman, who suffers from tingling or pains in the calves, cramps and weakness of the legs

*On Examination:* The calves are tender on deep pressure, and areas of cutaneous anæsthesia may be found over the legs. In the early stages the knee-jerks are increased, later they are diminished or absent. The leg muscles are weak and may be wasted. In some cases, in addition to the neuritis, there are cerebral symptoms which constitute Korsakow's psychosis. The main features are disorientation as regards time and place, the patient is unable to remember events which have recently happened such as what she had at her last meal or may say she has been out for a walk that morning when she has been in bed for several weeks. Variable oculo motor paralyses and a myotonic pupil (see p. 390) may be seen

*Cardio renal System* The patient complains of lassitude, headache, shortness of breath and perhaps swelling of the ankles

*On Examination* The heart shows signs of fatty or fibroid myocardial degeneration. The blood pressure is raised the arteries thickened and the urine contains albumin

*General Incapacity* There are other cases, especially in secret drinkers where no localising effects of the alcohol are present, but the patient is unfitted to a varying degree for his work. He is irritable, lacks concentration and his mentality somewhat resembles that of a child. He tries to give up the alcohol but has not sufficient will power

*Differential Diagnosis* The diagnosis is usually clear, but other drug addictions may have to be excluded. The condition of the pupils and knee jerks may suggest tabes dorsalis. The tender calves and absence of other signs of tabes serve to differentiate

*Course and Complications* The course is usually progressive, the patient becoming more and more a slave to alcohol. Delirium tremens (see p. 744) is liable to occur. Pulmonary tuberculosis may follow as a complication. Venereal disease may be contracted when under the influence of drink. According to Mott, chronic alcoholism is not an important cause of insanity, but probably accounts for one-fifth of

the cases of suicide, and for three-fifths of homicidal cases in England.

**Prognosis.** The outlook is always very grave.

**Treatment.** The patient should be admitted to an institution devoted to the cure of alcoholism and drug addiction. In order to prevent the onset of delirium tremens the alcohol should be "tapered" gradually, and not cut off at once. It is given in definite amounts at fixed intervals, such as every 2 to 6 hours. A reduction of about 2 oz. may be made every 24 hours. During this stage the patient is best in bed, and a sedative mixture can be given if required such as Sod. brom. gr. 30, tnc. capsici m. 3, aq. chlorof. ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  t.i.d.s. In some cases stronger sedatives are required, such as paraldehyde m. 30 to 60 once or twice daily. The use of vitamin B<sub>1</sub> in the treatment of alcoholic peripheral neuritis appears of very doubtful value. As soon as possible the patient should take a good mixed diet.

### Methyl Alcohol Poisoning

This is more likely to occur in countries where there is prohibition. The chief symptoms are giddiness, dyspnoea, nausea and vomiting. Optic atrophy is prone to ensue. It is a powerful poison and liable to cause death.

**Treatment.** The patient should be given 1 fl. oz. of castor oil and an intravenous injection of a pint of a 5% sodium bicarbonate solution to combat acidosis. The effect and dosage can be checked by the determination of the alkali reserve of the blood.

## THE ALCOHOLIC INSANITIES

### Delirium Tremens

Delirium tremens usually occurs in subjects of chronic alcoholism, either after an overdose or more commonly when their usual allowance is suddenly cut off, as may arise from the patient being put in prison or in a curative institution where the alcohol is suddenly withheld. Shock, such as an accident and a toxic disease, especially lobar pneumonia, when associated with withdrawal of alcohol, are important causes. The delirium begins with nocturnal visual hallucinations. Objects, which usually appear blue, such as snakes, rats or other animals, are imagined crawling on the bed or about the room. The patient is alarmed and often difficult to restrain. Auditory hallucinations of threatening voices are also heard.

**Differential Diagnosis.** The onset of delirium tremens may closely resemble that of uræmia owing to the presence of convulsions, oliguria, albuminuria and urinary casts. The blood nitrogen figures are, however, usually normal, but may be raised. The temperature may be normal or raised. The condition usually passes off in 2 to 3 days, the patient sleeping soundly; he may, however, pass into a state of muttering delirium which is followed by death.

**Treatment.** The patient should be kept in bed in a darkened room, and restrained if violent. The bowels should be opened with calomel gr. 5, or if he will not swallow this, a minim of croton oil in butter



should be placed on the tongue Saline purgatives should be given subsequently Sleep should be induced by giving m 60 to 120 of paraldehyde by mouth If this fails a hypodermic injection of hyoscin hydrobrom gr 1/100 should be given once or twice a day If the patient is difficult to restrain an injection of apomorphin hydrochlor gr 1/10 and strychnin hydrochlor gr 1/30 will sometimes quieten him It is usually necessary to give 1 to 2 oz of brandy or whisky a day for 2 or 3 days, which is gradually reduced to nothing The patient should be encouraged to take as much fluid as possible especially milk

### Mania a Potu

#### (Pathological Inebriation)

An individual so predisposed shows maniacal homicidal or suicidal tendencies after taking a small dose of alcohol Recovery is rapid if no more alcohol is taken

### Dipsomania

There are recurrent attacks of acute alcoholism but in the intervals the patient may be a teetotaler

### Alcoholic Automatism

A patient who is under the influence of alcohol may act automatically and apparently naturally for hours and days, having no recollection subsequently of what has occurred

### Alcoholic Pseudoparesis

This resembles the onset of general paralysis of the insane There are tremors of the tongue and hands, mental confusion an ataxic gait and epileptiform convulsions may occur

### Chronic Hallucinatory Insanity

The patient suffers from delusions of persecution and may hear voices

### Alcoholic Paranoia

The patient has delusions but there are usually no hallucinations

### Alcoholic Dementia

This is the final stage of alcoholic affection of the brain The patient is irritable, suffers from delusions loss of memory and general weakness

### Benzol Poisoning

Etiology Benzol poisoning is an industrial disease which may occur in distillers of coal tar, manufacturers of benzol mixtures for motor cars in rubber manufacturers and in workers applying paint by the spray process

**Pathology.** The post-mortem appearances in acute benzol poisoning resemble those of asphyxia, with hyperæmia of the various organs.

**Clinical Findings. Acute Poisoning.** This results from inhalation of air containing a high concentration of benzol. The individual becomes dizzy and rapidly collapses and death may occur in a few hours.

**Subacute and Chronic Poisoning.** The subject becomes weak and suffers from headache; a severe and progressive anæmia of an aplastic type develops, in which there is a progressive diminution of both red and white cells. Severe hæmorrhages may occur into the skin, and from the nose, mouth, and intestinal and genito-urinary tracts.

**Differential Diagnosis.** The nature of the patient's occupation, the typical anæmia and leucopenia establish the diagnosis.

**Course and Complications.** The course is progressive, unless the source of the poison is removed.

**Prognosis.** This depends upon the concentration of benzol in the air inhaled.

**Treatment. Prophylactic.** In dangerous occupations adequate ventilation should be enforced, and the workers subjected to monthly blood counts.

**Curative.** A blood transfusion should be performed in all cases showing marked blood changes or hæmorrhages.

### Carbon Monoxide Poisoning

**Etiology.** Carbon monoxide is present to the extent of about 15% in coal gas. It is also found in fumes from anthracite or charcoal fires, in the exhaust gas of petrol engines, and in the gas ("after-damp") formed in explosions in coal mines. Carbon monoxide poisoning may thus occur as: 1. An industrial accident, in a coal mine explosion. 2. A home accident, from using an improperly ventilated bath geyser, gas-stove, anthracite or charcoal stove, or from the exhaust of a car, whose engine is kept running in a closed garage. 3. A means of suicide, from a gas-oven or gas-stove.

**Pathology.** Carbon monoxide combines with avidity with hæmoglobin in the blood, forming carboxy-hæmoglobin and displacing oxygen. Anoxemia thus ensues. Poisonous symptoms such as malaise and headache may be expected if the blood is 30% saturated with CO. Unconsciousness ensues with 50 to 55% saturation, and in fatal cases the blood is usually about 80% saturated. Some patients die from nervous lesions, even although all CO has been removed from the blood. Post-mortem: The face and lips are pink, the blood is cherry red and minute hæmorrhages may be found in the brain, lungs and other organs. There is usually œdema of the lungs. Bilateral degeneration of the globus pallidus (Kolisko's lesion) and cerebral œdema may be found.

**Clinical Findings. Acute Poisoning.** The patient is usually discovered unconscious, with stertorous breathing, frothy exudation on the lips, injection of the conjunctivæ, dilated and fixed pupils, a pink colour in the face and lips, a rapid pulse and low blood pressure.

**Chronic Poisoning.** The symptoms develop insidiously, the patient

probably not knowing that he is breathing a poisonous gas; although the carbon monoxide has a faint odour of garlic. He suffers from lassitude, headache, giddiness, palpitations and nausea, there is a failure of mental powers, the patient cannot read clearly, and then finds he is unable to move his limbs. Sensibility to pain is also abolished. There is usually no dyspnoea. 10 mls of blood should be removed from a vein, and placed in a small tube, corked, and sent to a laboratory for examination for the spectrum of carboxy hæmoglobin.

**Differential Diagnosis** The circumstances in which the patient has worked or is found usually give a good clue to the diagnosis, which is established by detection of carbon monoxide in the blood.

**Course and Complications** Death may rapidly ensue, but the course depends upon the degree of saturation of the blood with carbon monoxide. Bronchopneumonia and myocardial degeneration with premature systoles may occur as complications. Sequelæ include confusional psychoses and a Parkinsonian syndrome.

**Prognosis** This is very grave in acute cases unless the patient receives adequate treatment before the carbon monoxide in the blood rises over 50%.

**Treatment Prophylactic** Care should be taken that geysers and stoves have adequate flues and that motor cars are properly ventilated. In suspected atmospheres a canary in a cage forms a good danger signal, as it is very sensitive to carbon monoxide and will fall off its perch if exposed to small concentrations of the gas.

**Curative Acute Poisoning** The patient should be taken into the open air and artificial respiration given by the Schafer method. The patient is placed prone with a thick rug or coat under the lower ribs and epigastrium. The operator kneels across the patient's body, facing the head and places his hands on the lower ribs. The operator, by bending forward and throwing his weight on his arms, slowly presses on the lower ribs and so expels the air from the lungs. He then gradually relaxes his pressure by bringing his body upright again and allows the lungs to re-expand, without removing his hands from the patient's chest. This process is repeated twelve to fifteen times a minute. As soon as available, oxygen, or, better, oxygen containing 7% carbon dioxide should be administered for at least half an hour. It can be given through a nasal tube or nitrous-oxide mask and gas bag apparatus if necessary combined with artificial respiration. The Clausen head harness is useful for prolonged administration of oxygen and CO<sub>2</sub>, or if artificial respiration is required at the same time. If given by a nasal catheter 20% CO<sub>2</sub> in 80% O<sub>2</sub> is required to give a concentration of 7% CO<sub>2</sub> in the inspired air, with a flow of 5 to 10 litres a minute. The administration of CO<sub>2</sub> and oxygen combined will rid the blood of CO four times as quickly as will the inhalation of oxygen alone. In the meanwhile the patient must be kept warm and stimulants applied such as strychnin hydrochlor gr 1/30 and liq adrenal hydrochlor m 5 given subcutaneously, or 1.5 to 5 mls of Coramine (nikethamidum B.P. Add.) injected slowly intravenously. After apparent recovery the patient should be kept very still for several days, as movement may

cause heart failure. If there are signs of myocardial damage, the patient must be kept in bed for several months.

*Chronic Poisoning.* Recovery usually occurs rapidly if the patient is placed in a pure atmosphere.

### Poisoning by the Barbiturates

*Etiology.* The barbiturates or diureides include barbitone or Veronal (soluble barbitone), Medinal, Luminal, Luminal-Sodium, Dial, Phanodorm, Allonal, Veramon, Amytal, Pernocton, Evipan, Soneryl, Nembutal, etc. They are used as hypnotics, but in overdoses or owing to personal idiosyncrasies may produce coma.

*Clinical Findings.* In coma due to barbiturate poisoning the pupils are usually dilated and react to light (in very severe cases they are contracted and reactionless) and the tendon reflexes are often exaggerated. In some cases there is excited delirium. The patient may remain in coma for several days before death or recovery takes place. The temperature is high, apart from pulmonary complications, in severe cases of poisoning. Bronchopneumonia usually develops as a terminal event.

*Treatment. Prophylactic.* The Pharmacy and Poisons Act of 1933, which came into force in 1936, restricts the sale of these poisons, and they can now only be obtained on prescription.

*Curative.* The stomach should be washed out with 2 gallons of warm water, and sod. sulph. gr. 240 with ext. cascarr. sagrad. liq. m. 240 left in the stomach. If the bowels are not opened in a few hours an enema should be given, and 10 oz. of strong coffee subsequently injected into the rectum. Artificial respiration should be applied, and oxygen with 7% CO<sub>2</sub> administered, if there are signs of respiratory failure. Stimulant treatment should be given as soon as the stomach has been washed out. Picrotoxin is the most powerful analeptic drug for this purpose. It is given at first intravenously, using a 0.3% solution (1 mil. = 3 mg.), 6 to 12 mg. are injected according to the degree of coma present. If there is no improvement, as judged by slight movements, moaning or deeper respirations, the intravenous injection is repeated at intervals of 15 minutes. Slight twitching of the facial muscles indicates that a sufficiency of the picrotoxin has been given. The picrotoxin can be subsequently injected intramuscularly every  $\frac{1}{2}$  to 1 hour, when the optimum effect from the intravenous injections has been obtained. Pentothal Sodium (1 G. in 10 mls of distilled water) should always be at hand for intravenous injection, the dose being 0.3 G., should convulsions ensue from an overdose of picrotoxin. Further points in the treatment are concerned with the bed being warmed, and its foot tilted 6 to 12 inches on blocks, an airway should be maintained and pharyngeal secretions removed by suction. If the blood pressure falls below 80 mm. Hg. strophanthin gr. 1/200 should be injected intravenously, and if signs of pulmonary oedema appear Pituitrin (ext. pit. liq. B.P.) 1 mil. should be injected subcutaneously. The picrotoxin treatment described above appears superior to the use of large doses of strychnine, but if picrotoxin is not

available the strychnine treatment is as follows. An intravenous injection of strychnin sulph or nit gr  $\frac{1}{3}$  is given followed by gr  $\frac{1}{6}$  an hour later. Subsequent injections of gr  $\frac{1}{6}$  may be required every two hours for another five doses and every three hours for two doses. In some cases life has been saved after the intravenous injection of as much as gr 10 of strychnine during the course of two or three days. A careful watch must be kept for signs of strychnine poisoning which may occur while the patient is still unconscious. They consist of twitching of the masseter muscle on tapping over it, trismus or generalised muscle twitching. If this occurs the injections of strychnine should be stopped and an intravenous injection of 5 mls of Coramine (nikethamidum B.P. Add.) should be slowly given. As much as 70 mls of Coramine may be thus injected in three days. The strychnine injections may be subsequently repeated if the patient is still unconscious.

### Acute Morphine Poisoning

**Etiology** Morphine poisoning may result from an accident, an overdose being prescribed or dispensed or owing to idiosyncrasy on the part of the patient. It may also be taken in an overdose with suicidal intent.

**Clinical Findings** The patient is usually found comatose, with small inactive pupils, depressed reflexes, a cold and clammy skin, feeble pulse and weak respirations.

**Treatment** Even if the morphine has been injected, the stomach should be washed out, as it is excreted into the stomach. Two gallons of warm water should be used containing pot permang gr 60. Strychnin sulph gr  $\frac{1}{2}$  should be injected intravenously and further stimulants used if required such as  $\frac{1}{2}$  pint of strong hot coffee per rectum and Coramine (nikethamidum B.P. Add.) 5 mls injected slowly intravenously. To stimulate the respiratory centre oxygen, or oxygen and 7% CO<sub>2</sub> should be given as for carbon monoxide poisoning (see p 747) together with artificial respiration.

### Morphinism

(including Heroinism and Opium Addiction)

**Definition** Chronic poisoning from opium or its derivatives.

**Etiology** Morphine is taken by injection, heroin by injection or in a linctus or as snuff, opium by mouth or by smoking. **Predisposing causes** 1 Race. Opium indulgence is common in India, China, Persia and Turkey. 2 Age and sex. A habit is more easily established in women and in young people. 3 Disposition. Morphine addicts often have a psychopathic or neuropathic tendency, but only a small proportion are degenerates. 4 Pain and worry. The drug may have been taken to relieve sciatica, chronic rheumatism, asthma, bronchitis, gastric or duodenal ulcer, for over work or because of phobias of cancer or insanity. 5 Occupation. Doctors, nurses and pharmacists have greater facilities for access to these drugs. A morphine

habit is not usually established unless the drug is taken regularly for several weeks or months.

**Pathology.** There are no characteristic post-mortem appearances, and death is usually due to some intercurrent disease. Often in cases of acute poisoning no morphine can be recovered from the body, but in some instances it is present in the liver, kidneys, etc.

**Clinical Findings.** The patient may give a history that he began to take the drug to relieve pain, insomnia, or worry, and that he has been unable to discontinue it. He may be able to do good work and to play games on a fixed daily dose such as gr. 6 to 8 of morphine. He gains no pleasurable sensations from the drug, and wishes to give it up, but is unable to do so. In some cases there is no indication that the individual is taking drugs; in others, tremors or other symptoms suggestive of drug addiction may be noticed. In more advanced cases the patient's friends can give more information. They will testify to a change in character and habits. The addict tends to be secretive, loses concentration and application, is irritable and depressed, except when under the influence of the drug. He becomes careless in his habits as regards cleanliness and dress. In some cases as much as gr. 40 of morphine are taken daily.

**On Examination:** The patient may deny taking drugs, or make a frank confession. He may complain of alternating constipation and diarrhoea. The nutrition is usually poor, the complexion sallow, the hands moist and the nails show trophic changes. The pupils are usually small; they may be unequal. The tongue is furred and the breath is offensive. The pulse and respirations are slow. The deep reflexes are depressed, and patchy areas of hyperæsthesia may be present, especially on the soles of the feet. The sexual functions are depressed, the urine may contain a trace of albumin. The skin usually shows the marks of hypodermic injections and of old abscesses.

**Differential Diagnosis.** Whether or not a suspected person is a drug addict can readily be decided by isolating him, so that he is unable to obtain a supply. In an addict, symptoms of deprivation appear in a few hours. There is running from the eyes, yawning, sneezing and restlessness. In more severe cases the addict then becomes weak and trembling and suffers great agony. He may have abdominal pain, vomiting, diarrhoea, and collapse, and become maniacal or comatose and finally die.

**Course and Complications.** The course is usually progressive, as the addict is unable to break the habit, and gradually requires larger doses. He will resort to any subterfuge to obtain it. Complications include intercurrent diseases, such as septicæmia or pneumonia.

**Prognosis.** The addict cannot cure himself. The prognosis depends upon the duration of the habit, the adequacy of the treatment and the underlying cause. If the latter can be removed, the chance of a permanent cure is greatly enhanced.

**Treatment. Prophylactic.** The Dangerous Drug Acts of 1920-23 have made it more difficult for these habit-forming drugs to be obtained,

and they should not be prescribed unless the patient will only need them for a short time, or to relieve suffering in cases of incurable disease.

**Curative** Patients should be treated in special institutions by physicians skilled in the appropriate methods. Sudden withdrawal of the drug is still the best method of treating a young person who has only been an addict for a short time. The acute withdrawal symptoms usually subside after 4 to 5 days. During this time sedatives should be administered such as Luminal (phenobarbitonum BP) gr  $\frac{1}{2}$  to 1 2 or 3 times a day. Hot baths and massage are also helpful adjuncts. In cases of long standing addiction gradual withdrawal is usually necessary. The morphine is reduced in stages, injections being given at regular intervals, the patient not knowing what dose he is receiving or when injections of saline are finally substituted for the drug. At the same time an increasing tolerance for belladonna or atropine is established. Sleep is secured by increasing doses of Luminal at night. Psychological methods form an essential part of the treatment.

### Strychnine Poisoning

**Etiology** Strychnine poisoning is rare. It may occur accidentally or be due to attempted murder.

**Clinical Findings** The patient may notice a bitter taste on swallowing the fluid containing strychnine. Symptoms of poisoning rapidly ensue, muscular twitching passing into violent clonic and tonic convulsions. These may result in rupture of muscles or in opisthotonus, emprosthotonus or pleurosthotonus. The spasms pass off in a minute or so, and complete flaccidity ensues between successive fits. The pulse is feeble and frequent, and respiration may be interfered with causing asphyxia. The mind remains perfectly clear and the dilated pupils and staring eyes express the torture of the individual. Death is not usually delayed in fatal cases for more than an hour or so, and may occur in a few minutes.

**Treatment** Powerful sedatives should immediately be administered such as a hypodermic injection of morphin hydrochlor gr  $\frac{1}{4}$  and the injection of Pernocton or anæsthetisation with chloroform. If possible Pernocton should be given intravenously, 1 mil a minute of a 10% aqueous solution until the patient falls asleep. The average dose required varies from 2 to 8 mils. If owing to the convulsions it is impossible to give an intravenous injection, Pernocton should be injected intramuscularly, 1 mil per 20 lb of body weight. The weight of the patient can usually be sufficiently accurately guessed to within a stone. When the patient is under the influence of Pernocton or chloroform the stomach should be washed out with 2 gallons of warm water. If there are signs of respiratory failure artificial respiration should be given together with inhalations of oxygen and 7% CO<sub>2</sub>. When the effect of the Pernocton wears off a second dose can be given.

### Acute Cocaine Poisoning

**Etiology** Poisoning may occur accidentally as the result of an injection of a 10% solution of cocaine prepared for the anæsthetisation

of a mucous membrane, or from swallowing cocaine from a nasopharyngeal plug. Suicidal poisoning with death may result from an adult swallowing gr. 34 of cocaine.

**Clinical Findings.** After taking a poisonous dose by mouth the patient notices dryness and burning in the mouth, buzzing in the ears, palpitations of the heart, hammering in the head and general trembling. There is headache, the vision becomes impaired, and cramps occur in the muscles. The patient dies in about an hour.

**On Examination :** The pupils are widely dilated and the patient is pale.

**Treatment.** The stomach should be washed out with 2 gallons of warm water containing pot. permang. gr. 60. One mil. of Cardiazol (leptazolium B.P.Add.) should be injected intramuscularly immediately, and repeated in a quarter of an hour. Lobelin. hydrochlor. mg. 10 in 1 mil. should be injected intravenously for respiratory collapse.

### *Chronic Cocaine Poisoning*

**Etiology.** The cocaine is usually taken as snuff, or injected hypodermically, or chewed in the form of coca leaves. The drug is taken for its stimulating effect. Morphine and alcohol addicts may ultimately resort to it.

**Pathology.** Post-mortem, the liver, kidneys, spleen and lungs are congested, but there is no pathognomonic sign of cocaineism.

**Clinical Findings.** The victim derives a temporary sense of exhilaration followed by depression. As the habit is established the craving for the drug becomes irresistible. The addict is gradually unfitted for mental or physical work, loses his appetite, suffers from insomnia and his muscles are weak and tremulous. Hallucinations develop, and irritation of the skin giving rise to the sensation of creeping insects (cocaine bugs). This is felt chiefly on the palms. There may also be delusions of persecution. Sexual excitement is increased but sexual power is diminished.

**On Examination :** The pupils are usually dilated and the pulse frequent. The nostrils may be inflamed, and acne occur on the face near the nose.

**Differential Diagnosis.** The general appearance of the patient suggests that he is taking drugs, the nature of which can only be found definitely by close observation.

**Course and Complications.** The habit is usually progressive; the patient may become insane.

**Prognosis.** The outlook is unfavourable. There is often recurrence after treatment, and death may be due to an overdosage or suicide by some other means.

**Treatment. Prophylactic.** The Dangerous Drug Acts of 1920-23 restrict the sale of cocaine.

**Curative.** The patient should be sent to an institution for drug addicts where the treatment resembles that given for morphinism (see p. 751).



### Acute Atropine, Belladonna and Hyoscine Poisoning

**Etiology** Poisoning usually occurs accidentally in adults, as from swallowing a chest liniment in mistake for a cough mixture in the dark. A belladonna plaster may cause poisoning in some individuals. Eye lotions containing atropine may cause acute poisoning, especially in children. Children may also eat the berries of the deadly nightshade.

**Clinical Findings** The patient complains of burning and dryness of the mouth, dysphagia, nausea and diplopia. *On Examination* The patient may be found unconscious or in a state of excited delirium. The face is flushed and an erythematous rash may be present. The pupils are widely dilated and do not react to light. The pulse is frequent.

**Treatment** If the poison has been taken by mouth the stomach should be thoroughly washed out with 2 gallons of warm water, or an emetic given. A hypodermic injection of pilocarpine nitrate gr  $\frac{1}{2}$  should be given. If there is respiratory failure artificial respiration and the inhalation of oxygen, or of oxygen and  $\text{CO}_2$  should be given, as for CO poisoning (see p 747). Further stimulants should be administered, such as the slow intravenous injection of 5 mls of Coramine (nikethamidum B.P. Add.) and the rectal injection of 10 fl. oz. of strong hot coffee.

## FOOD POISONING

**Introductory** Ptomaine poisoning was the name formerly applied to poisoning by tainted food, especially tinned meat and fish. It was thought to be due to protein decomposition products produced by the action of micro-organisms. It is now believed that the majority of cases are due to infection with the micro-organisms themselves or their toxins, and rarely if ever to ptomaines.

### Meat Poisoning

**Etiology** Meat poisoning is usually due to infection with organisms of the Salmonella group, these include the B. enteritidis (Gaertner), B. cholerae suis, B. aertrycke and B. paratyphosus B. Less frequently staphylococci are the infecting organisms in canned meat. The meat may be infected by the hands of "carriers" of the organism or contaminated by the excreta of animals such as rats and mice. The meat is not usually altered in appearance or in smell, and toxins are not produced in the meat before ingestion. This is, however, not so in the case of botulinus infection, which is considered separately on p 755. Mutton rarely causes meat poisoning, veal, pork and beef being the chief offenders. Improper preservation of canned meat, insufficient cooking and delay in consumption of meat are predisposing causes of poisoning. Children and debilitated adults are more prone to infection, and outbreaks usually occur in hot weather. Other diseases which may be conveyed by meat include infection with worms, tuberculosis, and rarely with anthrax.

**Pathology** Post mortem hyperæmia may be seen in the mucous membrane of the small intestine, and to a lesser degree in the stomach.

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**Pathology** Post mortem, hyperaemia may be seen in the mucous membrane of the small intestine and to a lesser degree in the stomach.

**Incubation Period.** This is usually a matter of a few hours, but it may be prolonged to 2 or 3 days.

**Clinical Findings.** The onset is sudden, with shivering, nausea, vomiting and abdominal pain. If the offending food passes into the intestine, diarrhoea follows. The motions are at first loose and evil-smelling, and later they become watery and may contain mucus and blood.

**On Examination:** The tongue is furred, the temperature often raised to about  $101^{\circ}$  F. and an erythematous rash may appear. In severe cases the patient becomes collapsed and cramps occur in the legs. In some instances the causative organism can be recovered from the stools during the first few days, and a specific agglutination reaction is obtained with the patient's serum in the second week of the illness. The urine may show acetone bodies if there is prolonged vomiting. The vomit should be kept and examined for arsenic. A specimen of the food should be retained for bacteriological examination.

**Differential Diagnosis.** The nature of the illness is usually suggested by the fact that more than one person who has partaken of the same food is affected. Acute abdominal conditions, such as appendicitis, should be excluded.

**Course and Complications.** The illness passes off in a day or so in slight infections; in more severe cases the symptoms increase in intensity for several days. A tendency to gastro-enteritis may persist subsequently.

**Prognosis.** The vast majority of cases recover completely.

**Treatment. Prophylactic.** Due care must be taken in canning food as regards the purity of the contents, and the temperature to which they are exposed during the process. The cans must remain hermetically sealed. "Blown" tins should be rejected; the contents of a tin should all be eaten the day it is opened.

**Curative.** The patient should be in bed and kept warm. In the early stages the stomach should be emptied by an emetic of mustard and warm water (oz.  $\frac{1}{2}$  in fl. oz. 10), or the stomach washed out with warm normal saline solution. A dose of fl. oz.  $\frac{1}{2}$  of castor oil should then be given. If the diarrhoea persists after the bowels have been well opened an astringent mixture should be given such as Bism. carb. gr. 10, cret. gr. 15, tnc. catechu. m. 20, aq. menth. pip. dest. ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  t.d.s. a.c.

If there is much abdominal pain, hot flannels or turpentine stupes (see p. 143) should be applied, and tinct. opii m. 10 can be added to the astringent mixture. Collapse is treated by stimulants such as hot coffee (fl. oz. 10) per rectum. Acidosis and dehydration are best combated by rectal injections of 4 to 8 fl. oz. of normal saline containing 5% dextrose every 4 to 6 hours. The patient should only take small quantities of boiled water or albumin water during the acute stages; later the diet is increased by adding dextrose (oz. 4 in a pint of water) and citrated milk (gr. 2 to oz. 1) diluted to half strength with water, rusks, arrowroot, cornflour, jellies, custard, thin bread and butter, eggs and fish.

## Botulism

**Definition** An acute variety of food poisoning due to a specific bacillus

**Etiology** Botulism is caused by the *Clostridium botulinum* (*B. botulinus*), of which there are two types. Type A produces the more virulent toxin. The bacillus is a spore forming anaerobic organism occurring in the intestines of animals and in soil. It produces an exotoxin acting on the nervous system. This toxin is present in the infected food. Any animal or vegetable food may be contaminated, but such articles as sausages, potted meat, ham, fish and canned meats and vegetables are most commonly affected.

**Pathology** Post mortem, hæmorrhages may be seen in the brain and spinal cord. The heart muscle is soft and the lungs may show bronchopneumonia. The toxin appears to paralyse motor nerve endings.

**Incubation Period** This is only a matter of hours, as the toxin is present in the food.

**Clinical Findings** The patient is taken ill after eating some contaminated substance, and usually several people are affected who have partaken of the same food. He feels ill with headache and usually complains of eye symptoms such as diplopia or blurring of the vision. The mouth is dry and there is dysphagia and often nasal regurgitation of liquids. The bowels are constipated, the legs and arms are weak, but there is usually no pain and the mind is quite clear. The voice may become very weak.

**On Examination** The pupils are dilated and do not react. Squint or nystagmus may be present and there is weakness of some of the external ocular muscles supplied by the third cranial nerve. There may be bilateral ptosis and paralysis of the palate. The tongue is furred and the temperature is usually subnormal. The pulse is slow at the onset but becomes more frequent later. There is flaccid paresis of the extremities and the deep reflexes are diminished. No sensory changes are present. The blood and cerebro-spinal fluid are normal and the causative organism is not usually found in the stools.

**Differential Diagnosis** The symptoms of botulism are somewhat analogous to those of belladonna poisoning. If the infected food is given to chickens they develop paralysis. Other forms of food poisoning such as those due to infection with bacteria of the salmonella group (see p. 753) must be excluded. In the latter the chief symptoms are gastro-intestinal. There are no muscular pareses, no visual disturbance, and the temperature is usually raised.

**Course and Complications** The acute stage of the disease is usually short, lasting 3 or 4 days. Convalescence, however, is slow, and disturbances of vision may persist for several weeks.

**Prognosis** The mortality rate is high, usually over 70%, and the shorter the incubation period the more likely is the disease to be fatal.

**Treatment Prophylactic** The bacilli and spores are killed by heat,

such as 120° C., for 6 minutes. Foods, when being canned, should therefore be heated to an adequate temperature.

**Curative.** The patient should be kept in bed and the stomach immediately washed out with warm normal saline. A polyvalent botulinus antitoxin, if obtainable, should be given in doses of 50 to 100 mls intramuscularly, or in severe cases 50 mls intravenously. If signs of respiratory failure occur, the patient should be placed in a Drinker respirator. An immediate injection of morphin. sulph. gr.  $\frac{3}{4}$  should be given, as this delays the action of the toxin.

### Diseases Carried by Milk

Milk forms a good culture medium for micro-organisms, and impure milk may cause much sickness. The diseases conveyed by milk include :

**Tuberculosis.** Infection comes from the cows, more rarely from human sources.

**Diarrhœa.** This is usually due to organisms such as Gaertner's bacillus and streptococci.

**Tonsillitis.** Streptococcal infection in milk may cause an epidemic of acute tonsillitis.

**Enterica Group Infections.** Milk is usually contaminated by the hands of a "carrier" or by infected water used for washing the cans or diluting the milk.

**Cholera and Dysentery.** The organisms usually gain access to the milk through infected water.

**Scarlet Fever.** Milk is probably infected from contact with milkers who have suffered from a mild unrecognised attack of scarlet fever, or who are carriers of the disease.

**Diphtheria.** Infection here is usually from carriers of the disease.

**Milk Fever and Abortus Fever.** This is conveyed by goat's and cow's milk.

**Foot and Mouth Disease.** Men may be affected by milk of cows suffering from the disease.

**Milk Sickness.** This is a disease which may affect man owing to consumption of the milk of cows suffering from "the trembles."

The characteristics of a milk epidemic : The onset is usually fairly definite and limited to a group of houses or a district supplied from one source. The occupants of houses in which the milk is boiled are usually spared. When scarlet fever or diphtheria is due to milk the symptoms are usually slight.

**Treatment. Prophylactic.** The purity of the milk supply should be guaranteed by Public Health measures. This involves inspection of cows and cowsheds, cleanliness in milking, pasteurisation, rapid distribution and the use of sealed bottles. In very hot weather milk should be boiled directly it is received in the house, and all milk given to infants, unless adequately pasteurised, should be brought to the boil to lessen the risk of infection with tuberculosis. The various grades of milk available are described on p. 155.

*Curative* This is considered in the sections dealing with the various diseases

### Fish Poisoning

Fish may be contaminated with the salmonella group of organisms. Oysters are liable to infection also with the enterica group. Mussels may cause poisonous symptoms owing to a toxin called mytilotoxin. *Diphyllobothrium latum* infestation may be caused by caviare and by fish such as the pike, carp, etc.

### Potato Poisoning

Sprouting potatoes may cause toxic symptoms. This may be due to a poison called solanin produced by the action of micro-organisms in the potato. There is headache, abdominal pain and gastro enteritis.

### Mushroom Poisoning

This is due to eating fungi which are mistaken for edible mushrooms. Death may occur in a day or so after severe gastro enteritis and hæmoglobinuric nephrosis.

### Cheese Poisoning

Infection with the salmonella group may occur, in some instances a toxin, tyro-toxicon, may develop in cheese.

### Rye Poisoning

The fungus *claviceps purpurea* may occur on rye or grains causing ergotism. Gangrene of the extremities or nervous lesions may develop.

### Lathyrism

Vetch seeds, if used as a substitute for wheat, may cause symptoms of poisoning. There is spastic paralysis of the legs with lumbar pain.

### Food Idiosyncrasies

Certain individuals are sensitive to special articles of food. Thus allergic symptoms may result from eating fish, eggs, milk, etc. An urticarial rash, nausea, vomiting, dyspnoea and collapse are the most prominent symptoms.

## POISON GASES

Poison gases used in war, may be classified as —

1 **Vesicants** *Mustard gas* is a yellow brown oily liquid, which penetrates clothes, rubber and wood and is neutralised by bleaching powder. It smells of mustard or garlic. Its action is insidious and its effects may not be noticed for several hours. It may be dispersed by aircraft spray, by bombs or by shells. The vapour or liquid will burn the skin especially in the flexures, and produce acute conjunctivitis,

blepharitis, laryngitis, tracheitis, bronchitis, bronchopneumonia and acute gastritis. It does not produce an immediate sense of irritation on the skin. The eyes, face and respiratory passages may be protected by a well-fitting respirator, but the remainder of the body is vulnerable.

**Treatment.** All contaminated clothing must be rapidly removed, liquid taken off the skin with cotton-wool, avoiding rubbing, the eyes washed out with warm 2% sod. bicarb. solution, and a drop or two of 2.5% Albucid Soluble instilled. Cocaine must not be used to relieve eye pain, as it will damage the cornea. The hair should be clipped short, and the body washed with warm alkaline soap and water. Anti-gas ointment No. 2 (chloramine-T in a vanishing cream basis) should be applied to all contaminated areas of skin, but it must not be used for areas which show erythema, nor for the eyes or scrotal region, owing to its irritant effect. Areas of cutaneous erythema should be dusted with equal parts of zinc oxide, talc, and boric acid. Blisters are pricked, and the surrounding skin cleaned with 20% Dettol. A pad of sterile gauze, soaked in sterile normal saline can be applied over blisters and raw areas, or gauze soaked in amyl salicylate, covered with cellophane, a thin layer of cotton wool and a bandage. Amyl salicylate must not be used for face burns as its vapour irritates the eyes. Later, gentian violet jelly or a crude neutral cod-liver oil dressing is used.

**Lewisite** (chlorovinyl-dichlorarsine), a colourless liquid, smelling of geraniums, is very pungent, and so more easily detected. It has great powers of penetration, and produces a rapid effect upon the eyes, skin and respiratory tract. It penetrates oilskins more easily than does mustard gas, but rubber and leather offer more resistance than they do to mustard gas.

**Treatment.** This is similar to that required for mustard gas. Hydrogen peroxide (20 vols.) applied to contaminated areas of skin will prevent vesication.

**2. Choking Gases.** These include *chlorine*, *chloropicrin*, *phosgene* and *di-phosgene*. Chlorine forms a greenish-yellow vapour smelling of bleaching powder. Chloropicrin smells like chlorine, but is more irritant. Phosgene is a colourless gas, smelling of musty hay, and di-phosgene has a similar smell. These gases produce cough, lachrymation, pain in the chest, dyspnoea, cyanosis (blue, or, in the most grave cases, grey), and oedema of the lungs. The effect of phosgene may not show itself for 24 to 48 hours.

**Treatment.** The respirator affords adequate protection. The patient must be kept warm, and at rest in bed. Cyanosis is treated by venesection and the administration of oxygen. Atropine and morphine are best avoided.

**3. Paralysing Gases.** *Hydrocyanic acid* smells of bitter almonds, and *hydrogen sulphide* of rotten eggs. They paralyse the respiratory centre, producing vertigo, coma and convulsions.

**Treatment.** The respirator gives efficient protection. Artificial respiration should be given, and oxygen with 7% CO<sub>2</sub> inhaled.

**4. Lethal Gases.** *Arseniuretted hydrogen* is odourless when in dilute concentration, and can be recognised by its property of turning



mercuric chloride test papers a yellow or orange colour. It produces weakness, headache, vomiting, hæmoglobinuria, anæmia and jaundice.

**Treatment** Sod citras gr 180 to 240 should be given daily to render the urine alkaline and aid diuresis. Plenty of fluids should be drunk. Dextrose should be given by mouth, and anæmia treated by a blood transfusion of 500 mls. Oxygen is required if the anæmia is severe.

**5 Lachrymators** *Ethylchloroacetate* is a dark brown liquid with a fruity smell, *bromobenzylcyanide* is a brown pungent liquid, and *chloracetophenone* smells of "Ronuk". They produce tears and blepharospasm, and recovery is usually rapid without treatment.

**6 Nasal Irritants**, such as *diphenylaminechlorarsine* and *diphenylcyanoarsine*. These produce sneezing, aching in the chest, throat and nose, and mental depression. The effects pass off rapidly in fresh air.

**7. Accidental Gases** *Carbon monoxide* may be liberated in badly ventilated "tanks," "pill boxes" and gun emplacements, or in mines, tunnels, burning buildings or in badly ventilated spaces warmed by charcoal braziers, etc. The ordinary respirator gives no protection. *Nitrous fumes* are evolved by burning cordite, and produce effects resembling those of phosgene. Partial protection is afforded by the respirator. *Screening smokes* may contain phosphorus chlorosulphonic acid, etc.

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mixture, diluted with water and drunk effervescent. In addition to the quinine, plenty of fluid should be taken. In malignant tertian infections of a pernicious type, or when many parasites are present in the blood, a rapid quinine effect is produced by intravenous injection of quinin. dihydrochlor. gr. 10 dissolved in 20 mils of sterile water. To prevent shock the injection should be made slowly. This may be repeated in 6 hours if the condition has not improved. Intramuscular injections are not advised, as they cause local muscular necrosis, and may be followed by abscess or tetanus.

Plasmoquine (pamaquina B.P. Add.) is a synthetic quinoline substance which destroys malarial crescents. Tablets containing 0.01 G. or 0.02 G. are given t.i.d. p.c. for 5 days. Toxic symptoms such as methæmoglobincythæmia, cardiac irregularities and abdominal pains may ensue. Its effect is better when administered with quinine as in the form of Plasmoquine Compound which contains plasmoquine 0.01 G. and quinin. sulph. 0.125 G. The usual dose is 2 tablets b.i.d. p.c. for 7 days. The chief use of plasmoquine is to diminish the number of relapses. Atebrin (mepacrine hydrochloridum B.P. Add.) is also of value in the treatment of malaria. The chief indications for its use are quinine intolerance, pregnancy and blackwater fever. It acts on intracellular asexual parasites in the red cells. The usual dose is 1 tablet (0.1 G.) crushed in water directly after meals t.i.d. for 5 days.

The cachexia which is liable to follow recurrent attacks of malaria is best treated by arsenic and iron, as in the following prescription. Ferri et ammon. cit. gr. 5, liq. arsenical. m. 2, tnc. nuc. vom. m. 5, sp. chlorof. m. 7, aq. ad fl. oz.  $\frac{1}{2}$ . Fl. oz.  $\frac{1}{2}$  ex aqua t.d.s. p.c.

### Blackwater Fever

#### (Malarial Hæmoglobinuria)

**Definition.** A complication of malaria, characterised by hæmoglobinuria.

**Etiology.** Blackwater fever is usually considered to result from chronic infection with subtertian malaria. A chill, fatigue, a dose of quinine, pamaquin or mepacrine hydrochloride may precipitate an attack. The destruction of the red cells is presumably due to a lysin, but no such lysin has been demonstrated in the peripheral blood. Parasites are found in the blood in over 70% of cases before the onset of the hæmoglobinuria, usually of the subtertian type. When severe hæmolysis is taking place the parasites are difficult to demonstrate in the blood. *Predisposing causes:* 1. *Locality:* Districts where the incidence of subtertian malaria is high and the disease is endemic. It is thus especially prevalent in tropical Africa and in certain parts of India, etc. It occurs in England in patients who have returned after contracting subtertian malaria abroad. 2. *Nationality:* Europeans are more prone than natives. 3. *Repeated attacks of malaria:* The patient has usually lived over 6 months in the tropical country. 4. *A previous attack of blackwater fever.*

**Pathology.** The kidneys are enlarged and very congested. The

renal tubules are obstructed with *débris*. The spleen and liver are enlarged, soft and pigmented. The brain and bone marrow may be pigmented and the heart show fatty changes. The hæmolysis is believed to occur in the general circulation and not in the kidneys.

**Clinical Findings.** The patient is usually an adult European, who has been in a malarial country for over 6 months and who gives a history of repeated attacks of malaria which have not been treated efficiently. The onset is generally sudden, with a rigor, the urine is then noticed to be red, and there may be some frequency. Feverish symptoms, such as malaise, thirst, headache and loss of appetite are complained of. There may be severe pain in the epigastrium or loins, with nausea, or vomiting of bile-stained fluid. Hiccough is a symptom in very severe cases.

**On Examination.** The patient is often jaundiced and the temperature is raised to  $102^{\circ}\text{F}$  or over. The spleen is palpable and tenderness may be elicited over it, in the epigastrium or renal areas. The urine. In some cases there is polyuria, in others oliguria or anuria. The urine varies in colour from almost black to pale red. A dark deposit settles on standing. The specific gravity is raised, and the reaction is acid. Albumin is present in considerable quantities. The red colour is due to blood. Met Hb being present and Oxy Hb in the severe cases. The deposit consists of blood and granular casts, epithelial cells and a few red cells. The blood. There is a hæmolytic anæmia of varying degree. The red cells may be diminished by 50% within 24 hours and may show ghost forms and punctate basophilia. The fragility of the red cells is normal. The white cells. The large mononuclears may show an increase up to 10% or more. The indirect van den Bergh reaction is positive and in severe cases the direct reaction is also positive. The blood urea is raised. Oxyhæmoglobin and methæmalbumin are present in the plasma.

**Differential Diagnosis.** The jaundice, temperature and bilious vomiting with dark urine are suggestive of yellow fever (see p 696). In yellow fever there is hæmatemesis and the urine contains bile. Other causes of hæmoglobinuria (see p 444) rarely require elimination. There is usually no difficulty in diagnosis.

**Course and Complications.** The hæmoglobinuria persists for a few hours or days, and relapses are not infrequent. The temperature falls to normal when the urine clears. Complications include suppression of urine and hyperpyrexia. Cholelithiasis may ensue as a sequela.

**Prognosis.** There are many mild cases which recover, but the outlook is very grave in any severe case.

**Treatment Prophylactic.** In districts where blackwater fever is endemic, every attack of malaria should be adequately treated with quinine. If a person has suffered from hæmoglobinuria previously, mepacrine hydrochlor should be used rather than quinine in the treatment of an attack of malaria, and the urine should be kept alkaline.

**Curative.** The disease should never be treated lightly. The patient must be put to bed and kept lying down at absolute rest. He should be given fluids containing dextrose as for acute nephritis (see p 449) the

amount of urine passed must be measured and charted daily, and when diuresis occurs the volume of ingested fluid can be increased. An alkaline mixture containing Sod. bicarb. gr. 30, sod. citrat. gr. 20, sp. chlorof. m. 7, aq. menth. pip. dest. ad fl. oz. 1 should be given six-hourly or four-hourly, to render and keep the urine alkaline. If vomiting prevents this, drip rectal salines, containing sod. bicarb. gr. 120 to 1 pint, should be given. If there is suppression of urine, the loins should be dry cupped, or hot applications, such as water bottles, applied. An intravenous injection of 1 pint of 5% dextrose in normal saline may prove helpful. If vomiting prevents the taking of sufficient fluid, rectal injections of 4 to 8 fl. oz. of normal saline containing 5% dextrose should be given every 4 to 6 hours. The bowels are opened with an enema if there is constipation, before the rectal salines are given. A drip blood transfusion of 500 to 1,000 mils should be given to patients with polyuria. For peripheral circulatory failure, treatment consists of warmth, the intramuscular injection of 1 mil. of Pitressin and the oral administration of ephedrine hydrochlor. gr.  $\frac{1}{2}$ , repeated 6 hourly. It is usually considered inadvisable to prescribe any anti-malarial drug during the acute stages of the illness, and the anaemia generally improves spontaneously during convalescence. After recovery it is advisable for the patient to leave a malarial country, as dangerous relapses are to be feared. If parasites reappear, as often happens five to fourteen days after the haemoglobinuria has ceased, a course of Atebrin (mepacrinæ hydrochloridum B.P. Add.) should be given (see p. 682).

### Bacillary Dysentery (Epidemic Dysentery)

**Definition.** A disease characterised by diarrhoea with the passage of blood and mucus, and caused by a special group of bacilli.

**Etiology.** There are three main types of bacilli: The Shiga (including Schmitz's bacillus), the Flexner-Y and the Sonné bacillus. There are several strains of the Flexner group distinguishable serologically. The Shiga bacillus forms exotoxins affecting the central nervous system and endotoxins acting locally on the intestinal mucous membrane. Flexner and Sonné infections are generally of a milder type than are those caused by the Shiga group. Infection is carried to man by food and water, and transmitted from faeces by flies or by fingers. *Predisposing causes:* 1. *Locality:* Bacillary dysentery occurs in epidemics in the sub-tropics and also in temperate climates. Outbreaks of dysentery in England, including summer diarrhoea in children, are most often due to the proteus *morganii*, *B. sonnei* and *B. enteritidis* (Gaertner). It affects armies, prisoners and lunatics in asylums. 2. *Season:* In the tropics during the rainy season. 3. *Debility.* 4. *Age:* Infants under 2 years and adults of either sex.

**Pathology.** The bacilli produce an acute inflammation of the mucous membrane of the large intestine, and the last part of the ileum may also be affected. Small superficial pinkish ulcers and larger irregular transversely disposed ulcers form, and there is no undermining

of their edges. Later the mucous membrane may necrose, becoming greenish black. This may slough and leave a firm rigid tube of bowel.

**Incubation Period** This varies from a few hours to about 7 days.

**Clinical Findings** The patient is usually an adult, who is suddenly taken ill with abdominal pain and diarrhoea. There is malaise associated with fever and tenesmus may be very marked. In an acute case the patient has to go to stool very frequently, but little is passed each time. There may also be vomiting and considerable thirst.

**On Examination** The abdomen is tender and the muscles are often held rather rigidly contracted. The temperature is raised to  $102^{\circ}\text{F}$  or  $103^{\circ}\text{F}$  and the pulse is frequent. The tongue in a severe case becomes dry and the patient suffers from marked collapse owing to dehydration and toxæmia. The urine output is scanty. The stools often consist of a little jelly like material with blood, but with no fecal matter, and they are odourless. Microscopically, a film shows very few bacilli, many polymorphonuclear cells (up to 90%), some large macrophage cells with engulfed red corpuscles (these must be distinguished from amœbæ). Dysentery bacilli may be isolated by culture during the first few days of the illness, but rarely after the first week. The blood. A positive agglutination with stock dysentery bacilli cultures can be obtained after the first week, most definitely in Shiga infections. Blood culture is rarely positive, there is usually a slight leucocytosis. In severe cases the blood urea rises and the alkali reserve falls.

**Varieties** 1 Fulminating dysentery. This may be of a choleraic type, with collapse, vomiting and diarrhoea or a gangrenous type, with severe toxæmia and abdominal pain. 2 Mild type. There is little constitutional disturbance or tenesmus and the stools contain fæces, with some mucus and blood. 3 Chronic type. This persists for over a month, often with alternating constipation and diarrhoea. 4 Infantile or summer diarrhoea. Diarrhoea with blood and mucus in the stools may be due to bacillary dysentery.

**Differential Diagnosis** Bacillary dysentery differs clinically from amœbic dysentery in its more acute onset, higher temperature, greater degree of tenesmus, and more profound collapse. The stools differ in their appearance, those of amœbic dysentery usually being very offensive and containing fæces. They differ also in their cellular exudate (see p. 687). The causative organism is also distinct. Arthritis is a complication of bacillary dysentery, and liver abscess of amœbic dysentery. Other conditions which can be excluded clinically and bacteriologically are the enterica group infections, food poisoning, cholera and shistosomiasis. Ulcerative colitis is in some cases due to bacillary dysentery (see p. 52). Sigmoidoscopic examination is of value in the diagnosis of chronic dysentery. The mucous membrane is red, granular and bleeds easily, and the bowel wall is rigid.

**Course and Complications** In fulminating cases the course is rapidly progressive to death. In acute cases the diarrhoea usually lasts for 7 to 10 days, and the condition gradually improves as the motions become feculant. Relapses may occur, the stools containing blood and

mucus and a condition of chronic dysentery becomes established. Complications include arthritis, parotitis, conjunctivitis, iridocyclitis, ascites and constriction of the intestine. The arthritis usually affects large joints such as the knees; there is clear fluid in the joint and recovery is the rule. It may occur during the acute stage or during convalescence.

**Prognosis.** The average mortality is about 2 to 5%. Fulminating cases die in a few days. *Shiga infections are more severe than other types.*

**Treatment. Prophylactic.** Endeavours should be made to prevent flies from having access to food; faeces should be disposed of hygienically; dysentery bacilli carriers should not be allowed to prepare food. Inoculation with dysentery vaccines has not been very successful.

**Curative.** The patient must be kept warm in bed. Hot applications to the abdomen, such as turpentine stupes, help to relieve pain. At the beginning of the illness the bowels should be emptied of irritant material by a dose of ol. ric. fl. oz.  $\frac{1}{2}$ . Then sodium sulphate, gr. 60 in 2 oz. of water should be given every 2 hours while the patient is awake, until watery evacuations are obtained. This tends to concentrate the toxins in the bowel and prevent their dissemination. The saline administration is then reduced to every 4 hours, and then to every 6 hours until faeces reappear in the stools. **The Diet:** During the first 24 hours only water or mineral waters should be allowed. Subsequently albumin water, dextrose water, thin arrowroot, Brand's essence and jelly are given. The diet is gradually increased by the addition of citrated milk, milk jelly, custard, sago pudding, a lightly boiled egg, toast or rusks, etc.

**Special Treatments.** 1. Antitoxic serum (polyvalent) has little effect if administered after the first few days of the disease. A preliminary determination of susceptibility to serum and desensitisation, if necessary, is made (see p. 535). In an acute case 50 mils of serum, diluted with an equal volume of warm normal saline (temperature 100° F.) are injected slowly intravenously twice a day until the temperature falls to normal. 2. Bacteriophage is strongly recommended by certain authorities. It must be administered during the first 2 or 3 days of the disease, combined with adequate rest and dietetic restrictions. 3. The use of Sulphaguanidine is a promising method of treatment. The usual dosage is 3 G. t.i.d. by mouth for 2 days, followed by 2 G. b.i.d. for 5 days.

If there is severe dehydration intravenous hypertonic saline should be given (see p. 691) as for cholera. Tenesmus can be relieved by a small starch and opium enema (Tnc. opii m. 20, starch. gr. 60, water fl. oz. 2). A subcutaneous injection of morphin. sulph. gr.  $\frac{1}{2}$  may be required for insomnia. In chronic cases which will not heal, rectal injections of Yatren (cliniosonum B.P. Add.), as for amœbic dysentery (see p. 689) may be tried. If these fail a cœcostomy will give rest to the large intestine and also enable it to be washed out, and is successful in some cases.



The arthritis is best treated by the application of Scott's dressing (ung hydrarg co B P)

### Amœbiasis

(*Amœbic Dysentery Amœbic Hepatitis Pulmonary Amœbiasis*)

**Definition** Amœbiasis includes the diseases in man caused by infection with the *Entamœba histolytica*

**Etiology** The causative agent is the *Entamœba histolytica*. This exists in an amœboid or vegetative form and as a cyst. The amœba is actively motile, has a clear ectoplasm and usually contains engulfed red cells. The cyst contains four nuclei. Man swallows cysts in food or water. In the intestine amœbæ develop, some of these amœbæ form cysts which are excreted in the fæces. The house fly probably carries the cysts to food and water, or food may be contaminated by the fingers of a cyst passer. **Predisposing causes** Locality, especially Egypt, India, Mesopotamia and parts of America.

**Pathology** The amœbæ infect the large intestine, working their way through the mucous membrane. They cause colliquative necrosis in the submucous tissues and a bottle shaped ulcer forms. The ulcers enlarge and have an undermined edge. They are situated longitudinally in the intestine, and form especially in the cæcum and at intestinal flexures. The amœbæ may pass to the liver *via* the portal vein and produce hepatitis, or single or multiple abscesses, the pus is pinkish brown (anchovy sauce) and usually sterile. Amœbæ are found in the scrapings from the abscess wall. The liver abscess may rupture into the lung, stomach, duodenum, colon, peritoneum, and rarely into the pericardium. Abscesses may form in the brain or spleen as the result of a systemic infection.

**Incubation Period** In experimentally infected man this varies between 9 to 94 days.

**Clinical Findings** The patient may be a child or an adult, who complains of diarrhœa coming on gradually, with abdominal pain. There is tenesmus if the lesions are situated low in the rectum.

**On Examination** Tenderness may be elicited over the cæcum or transverse colon or in the left iliac region. The temperature is usually not raised or only slightly so. In this acute simple type the bowels are opened about 12 times in the 24 hours and the motions contain mucus, pus, dark blood and some fæces. If untreated the condition usually remits after a few weeks, but recurrences are common. Examination of the stools. The specimen should be sent to the laboratory directly it is passed and examined for amœbæ or cysts, and if not found another specimen sent daily for 5 to 7 days. The cellular content of the stools in amœbic dysentery shows a preponderance of mononuclear leucocytes or degenerating epithelial cells. The field usually swarms with bacteria, and Charcot Leyden crystals are often present. In chronic cases diagnosis may at times be made by sigmoidoscopy and finding amœbæ in scrapings from an ulcer wall.

**Other varieties** include 1 *Acute gangrenous dysentery*. A short fulminating illness, in which hæmorrhage and perforation are liable to

mortality has been much lowered by closed aspiration treatment. Emetine hydrochloride gr. 1 in 1 oz. distilled water may be injected into the abscess cavity after aspiration.

### Cholera

**Definition.** A disease due to a specific bacillus, characterised by severe watery evacuations, muscular cramps and collapse.

**Etiology.** The cause is the *Vibrio cholerae* (comma bacillus). It is conveyed from man to man by means of infected water and food, such as melons, milk, etc. The vibrios pass out in the faeces and the water is thus contaminated. Fingers and flies may also convey the vibrios. Carriers occur, but they are usually convalescent patients who remain carriers for a few weeks or months only. The disease is disseminated largely by pilgrims or traders. *Predisposing causes:* 1. *Locality:* Cholera occurs chiefly in India, endemically and epidemically. Epidemics have arisen in Europe and America. 2. *Season and climate:* The incidence is favoured by a high absolute humidity of the air (over 0.400).

**Pathology.** At autopsy the body is wasted and dry; the muscles are dark. The chief lesion is in the ileum, where the mucous membrane is reddened and the contents may be of the "rice-water" character; white flakes of cellular *débris* in a clear fluid. The spleen is not enlarged, the kidneys may be congested or show cloudy swelling.

Cholera vibrios are usually confined to the intestine, but some may be found in the gall-bladder; more rarely there is a septicæmia, and they are present in the spleen and urine.

**Incubation Period.** This varies from a few hours to 5 or 6 days.

**Clinical Findings.** Cholera may begin quite suddenly, or there may be a few days of diarrhœa, not of a typical choleraic nature at the onset. The patient complains of very severe diarrhœa, in which after the bowel has been emptied of fecal matter, the evacuations consist mainly of water. There is marked prostration, and severe cramps in the legs and abdomen, which may cause muscle rupture. Vomiting of watery fluid adds to the patient's misery, and there is intense thirst.

**On Examination:** In this algid stage the skin is cold and clammy, and has lost its elasticity, the patient is cyanosed, the eyes are sunken, the respirations and pulse rate are rapid, and the voice is feeble. The axillary temperature is normal or subnormal, but in the rectum the temperature is slightly raised. The stools are of a "rice-water" character (see above) and contain cholera vibrios. Bacteriophage is present in stools of patients who do well, and not present in fatal cases. The urine becomes very scanty, with a high specific gravity and albumin is often present. The blood pressure falls below 100 mm. Hg. syst. The blood is "sticky" from concentration, the Hb. percentage rises, and the red cells number over 5,000,000 per c.mm. There is a leucocytosis of 20,000 to 50,000 per c.mm. The specific gravity of the blood rises from the normal of 1.050 to 1.061, if 1 pint of fluid has been drained away from the blood, and to 1.065 with a loss of 5 pints. The blood culture is usually sterile, but a positive agglutination of dead cholera vibrios by

the serum is found after the eighth to tenth days. The patient may rapidly die, or in a few days pass into the reaction stage. The body temperature rises, the skin becomes warm, the blood pressure rises, the pulse slows, and the output of urine increases, the watery vomit and stools cease. An irregular type of fever may now ensue with delirium and coma, called cholera typhoid, or the temperature may rapidly settle as the patient's condition improves.

**Varieties.** 1 *Cholera sicca*. The patient rapidly dies, the bowels contain much fluid, but no "rice-water" stools have been passed.

2 *Ambulatory*. Here the diarrhoea is not very severe, the constitutional disturbance is comparatively mild and there are no cramps.

**Differential Diagnosis.** There is little difficulty in an epidemic, and the diagnosis is made by examination of the stools for vibrios. Other causes of acute gastro-enteritis must be excluded, such as food poisoning, and in England cholera nostras or summer diarrhoea. In tropical countries algid malaria (see p. 680) is excluded by a blood film.

**Course and Complications.** The severe forms of cholera take a very rapid course, in less grave infections the course is more prolonged and relapses may occur during convalescence, with return of the diarrhoea. Complications include bronchopneumonia, parotitis, nephritis, uræmia and hyperpyrexia.

**Prognosis.** Cholera is a very severe disease, but the mortality has been lowered by modern treatment. In any epidemic the case virulence becomes less severe towards its close. The amount of fluid lost from the blood (as judged by the specific gravity) is a guide to the severity of the case.

**Treatment.** *Prophylactic.* In an epidemic, protection is afforded by boiling all water and milk, eating only cooked foods, avoiding all foods liable to cause diarrhoea, and by the isolation of contacts. Further, at any time vaccination will afford considerable protection for several months. The anti-cholera vaccine contains 1,000 million dead vibrios in 1 ml, the initial dose is 1 ml followed by 2 mls a week later.

*Curative.* At the onset of the diarrhoea the patient should be kept warm in bed. The fæces and urine should be received in disinfectant as for enteric fever. Morphine and opium should never be used. The patient should drink water, albumin or dextrose water, in small quantities frequently, taking as much as he can. Vomiting may be checked by giving tab. cocain gr  $\frac{1}{20}$  by mouth, repeated in half an hour if necessary. Rogers' hypertonic saline should be given intravenously in all cases in which there is much loss of fluid from the blood, as judged by the specific gravity, or in which the blood pressure falls to 80 or 70 mm Hg syst. The chlorides combine with the cholera toxins in the blood and the product is excreted. The hypertonic solution prevents fluid passing from the blood to the bowel. Uræmic symptoms are checked by maintaining the alkalinity of the blood by intravenous injection of an alkaline solution. Rogers' hypertonic saline solution contains sod. chlorid. gr 120, calc. chlorid gr 4, water 1 pint, and his alkaline solution contains sod. bicarb gr 100, sod. chlorid gr 90, water 1 pint. The solu-

tions should be warmed to body temperature, but if the rectal temperature is over 100° F., the solutions should not be over 80° F., or hyperpyrexia may ensue. They should be run in at the rate of about 4 oz. a minute. The amount required is determined by the specific gravity of the blood, as described on p. 690. One pint of the alkaline solution is first injected, and then the requisite amount of fluid is made up by injecting the hypertonic solution. The alkaline solution should also be injected into the rectum, at first 10 oz. every 2 hours, and then every 4 hours until the output of urine exceeds 20 oz. a day. Calcium permanganate pills (gr. 2) should be given every quarter of an hour for the first 4 hours and then every half hour, until green bile appears in the stools. The permanganate oxidises the toxins. Kaolin oz. 7, in water oz. 14, can be taken frequently and appears to do good. Other forms of treatment, such as the administration of essential oils by mouth, are not of value in severe cases. The use of antitoxic serum is still in the experimental stage. During convalescence the diet must be very cautiously increased with diluted citrated milk, milk jellies, custards, etc.

### Sprue

(*Psilosis*)

**Definition.** A disease characterised by emaciation, sore tongue, and diarrhoea.

**Etiology.** The cause is unknown. Sprue may be due to: 1. A deficiency of ionised calcium in the blood. 2. Infection with yeasts, such as the *Monilia psilosis*, or with streptococci. These are probably secondary infections. 3. Deficiency of the vitamin complex B in the food. *Predisposing causes*: 1. *Locality*: Especially China, the Malayan Archipelago, India, Ceylon, the East and West Indies, and the southern states of N. America. Sprue may occur in certain houses. 2. *Race*: Europeans are liable to sprue in endemic zones and also may develop it several years after returning home. 3. *Age and sex*: Chiefly adults and slightly more common in women.

**Pathology.** The wall of the small intestine is very thin, and the mucous membrane is atrophied. Small ulcers may form and perforate. The large intestine may be similarly affected. The heart, liver and spleen are atrophied and the bone marrow shows megaloblastic hyperplasia. The atrophied intestinal mucous membrane interferes with absorption.

**Clinical Findings.** The onset is insidious with weakness, dyspepsia and flatulence. Looseness of the bowels is then noticed, occurring only in the early part of the day. The patient loses weight progressively and complains of soreness of the tongue and mouth, and later soreness may also be referred to the œsophagus and rectum.

**On Examination**: The patient is very wasted when the disease is of some duration, and the skin is often dark. The tongue in the early stages is red, and later small vesicles may form and the surface is smooth and shiny. In more chronic cases the tongue is shrunken and pale. Erosions may also be seen on the buccal mucous membrane. The liver

dulness is diminished. A test meal often shows deficiency of hydrochloric acid. The stools are large, pale and frothy. Urobilinogen is present. The total fat is increased to 50 or 80% (normal 20 to 30% of dried faeces), there being an increase of the split fat. The blood shows often a megalocytic anaemia with a high colour index. A few normoblasts may be seen, but usually no megaloblasts. The leucocytes are usually not affected. The dextrose tolerance test often shows a low curve due to deficiency of absorption, and the blood calcium may be below normal. Sternal puncture shows changes similar to those found in pernicious anaemia (see p. 489).

**Differential Diagnosis.** The history of residence abroad and the clinical picture is characteristic. Corliac disease in adults (see p. 60) closely resembles and probably is identical with sprue. In chronic pancreatitis there is an excess of unsplit fat. In some cases the blood count may closely resemble that of pernicious anaemia.

'Hill diarrhoea' is liable to occur especially in India at an altitude of over 6000 feet. It resembles sprue with the pale, frothy stools occurring in the morning, and may develop into true sprue.

**Course and Complications.** The course is usually chronic. Haematemesis or tetany may occur as complications.

**Prognosis.** Death may occur within a year if the disease is not treated and in any case the prognosis is bad in patients past middle age.

**Treatment.** The patient must be put to bed and kept there for at least 6 weeks. The diet consists of milk, Benger's food or Yaghurt given in two hourly feeds, beginning with 3 pints of milk in the 24 hours and increasing gradually to 5 pints. It should be sipped very slowly. Sprue is rich in protein and poor in fat and is of value as an alternative to a milk diet. It is dissolved in water according to the directions and six feeds are given in the 24 hours at intervals of 2 hours, the total quantity taken being gradually increased from two to six pints. If milk food disagrees raw meat juice or 2 oz. feeds of lightly cooked minced steak may be given. The meat feeds are increased up to 1 to 2 lbs. daily. The motions should become formed after a few days' treatment. If constipation develops liquid paraffin should be given as required. After 6 weeks the diet should be gradually increased by adding fruit such as strawberries, bananas or apples 1 or 2 daily and gradually increasing to 1 lb. or more daily. A raw egg may be added to the milk, then 1 or 2 rusks, pounded fish and later chicken are given. Bism. salicyl gr 15 t.d.s. two hours after meals helps to check loose motions.

For the anaemia liver extract may be given as for pernicious anaemia (see p. 490). If the anaemia is very severe blood transfusion is of great value. Deficiency of ionised calcium is made good by calcium lactate gr 15 to 60 t.d.s. together with Radiostoleum capsules (liq. vitamin A et D conc. B.P. Add.) m 3 t.d.s.

After each feed the mouth should be cleansed with an alkaline solution containing sod. bicarb. gr 60 to 5 oz. of water. If the tongue is sore it can be painted with 0.2% cocaine solution before feeds. As soon as the patient is convalescent he should leave the affected area and not return.

## Plague

*(The Black Death)*

**Definition.** A disease characterised by septicæmia, and frequently by enlargement of a group of glands, due to infection with a specific bacillus.

**Etiology.** The cause is the *Pasteurella pestis* (*B. pestis*). Rats suffer from plague, and bubonic plague is conveyed to man by the bite of the rat flea (especially the *Xenopsylla cheopis*). Direct droplet infection from man to man causes the spread of pneumonic plague. *Pre-disposing causes*: 1. *Locality*: Plague is endemic in parts of India, Indo-China the East Indies, Siberia, North and East Africa, etc., and sporadic cases occur in Europe and in ports, such as London. In 1664-1665 the great epidemic occurred in London. 2. *Climate and season*: A mean temperature over 80° F. is unfavourable to plague. In temperate zones it occurs in the summer and autumn. 3. *Hygienic conditions*: Dirt and overcrowding favour its development. 4. *Age and sex*: No age or sex is exempt.

**Pathology.** The disease is a hæmorrhagic septicæmia. Post-mortem the body may appear livid (black death), owing to ecchymoses. The lymph glands at various sites are enlarged, matted together and surrounded with a hæmorrhagic œdema. Serous membranes show ecchymoses and blood-stained effusions. The spleen is enlarged and soft; the liver is enlarged, due to cloudy swelling, and small abscesses may be present; the kidneys may show cloudy swelling; ulcers may be found in the colon. The lungs may show hæmorrhagic broncho-pneumonic patches or lobar involvement. The causative organism may be isolated from the blood, lymph glands, spleen, lungs, etc.

**Incubation Period.** 2 to 10 days.

**Clinical Findings.** In bubonic plague the patient is suddenly taken ill with malaise, shivering, pains in the back and legs, nausea, and vomiting. He becomes very weak, is mentally confused and the gait may be staggering. In a day or so he feels a painful swelling in a groin or elsewhere, owing to the bubo.

**On Examination:** The face is dusky or flushed, the conjunctivæ injected and the expression may be wild. The temperature is raised, usually over 102° F., and the pulse frequent. The primary bubo is generally in the groin, the glands being enlarged, tender, and the subcutaneous tissues over them feeling œdematous. Less often the primary glandular enlargement occurs in the axilla or neck. Fluid obtained by gland puncture shows plague bacilli. The blood: There is a high leucocytosis, which may reach 80,000 per c.mm. The blood culture may show the *B. pestis*. The urine often contains albumin. The glands usually, but not invariably, suppurate in about 7 to 10 days. The temperature runs an irregular course, and in cases which recover it falls by lysis, when the glands, if they do not suppurate, diminish in size and often there is marked sweating. Skin lesions may occur, ecchymoses are common, but in addition there may be vesicles, pustules or areas of gangrene resembling carbuncles.

*Varieties* 1 *Abortive plague (Pestis minor)* Pyrexia may be slight or absent, and the enlarged glands are not very painful. Constitutional disturbance is slight and the patient remains ambulatory. The buboes may or may not suppurate.

2 *Pneumonic plague* This may be primary or complicate bubonic plague. The signs and symptoms are pulmonary, there is marked dyspnoea, with cough and expectoration.

*On Examination* The patient is dusky and cyanosed, râles may be heard scattered in the lungs, or areas of bronchial breathing with increased voice conduction. A pleural effusion often forms. The sputum is thin, watery and coloured pink with blood, it is not sticky and contains the *B. pestis*.

3 *Septicæmic plague* The patient usually dies before a bubo or bronchopneumonia has had time to be manifest.

4 *Intestinal plague* This is an unusual variety, in which buboes do not form, but there is diarrhoea with blood and vomiting. Plague bacilli are found in the stools.

5 *Cerebral plague* This is also a rare variety, characterised by convulsions and coma.

*Differential Diagnosis* In an epidemic there is little difficulty in recognising plague. Sporadic cases may be confused with other forms of septicæmia, with enteric fever, glandular fever, malaria (cerebral) or influenzal bronchopneumonia. Diagnosis is established by finding the causative organism in the fluid from gland punctures, the blood, urine, faeces or sputum.

*Course and Complications* The course of bubonic plague is as described above, the course may be rapidly fatal in the pneumonic, septicæmic or intestinal type, and this is the rule with cerebral plague. Complications include pneumonia in bubonic plague and severe hæmorrhage from erosion due to a sloughing bubo.

*Prognosis* The mortality for the pneumonic and septicæmic varieties is nearly 100%, the bubonic variety proves fatal in about 80% of cases in natives and in about 30% of cases in Europeans.

*Treatment* *Prophylactic* This is chiefly concerned with the destruction of rats, and prevention of rats from entering houses or ships. Nurses should wear masks in caring for cases of pneumonic plague. In an epidemic Haffkine's prophylactic vaccine (containing dead bacilli and toxins) should be given 1 000 millions for the first dose, and 2 000 millions 10 days later. It appears to exert definite protection.

*Curative* The patient should be isolated for 4 weeks after the temperature is normal. He must be kept in bed and the temperature lowered by sponging if it rises over 106° F. The buboes should be fomented every 4 hours with 1 in 4 000 perchloride of mercury solution. When the buboes suppurate they should be opened. An intravenous injection of 30 to 100 mls of Yersin's anti serum diluted with an equal volume of normal saline should be given and repeated daily if the temperature does not fall. Good results have been obtained by the use of Sulphathiazole (M & B 760) and Sulphapyridine (M & B 693). In